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

By

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A thesis submitted to the School of Pharmacy in partial fulfillment of the requirements for the degree of Bachelor of Pharmacy (B. Pharm.)

School of Pharmacy
Brac University
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Declaration

It is hereby declared that

1. The thesis submitted is my own original work while completing 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 Dhaka, Bangladesh" submitted by Adiba Sultana (19146004), of Summer, 2024 has been accepted as satisfactory in partial fulfillment of the requirement for the degree of Bachelor of Pharmacy.

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Ethics Statement

This project does not involve any kind of animal and human trial.

Abstract

The recent emergence of multidrug-resistant pathogens poses a significant threat to global public health. The major objective of this study was to investigate the prevalence and antimicrobial susceptibility patterns of clinical isolates collected at a selected hospital of Bangladesh. Out of 1890 samples, only 489 showed positive for pathogens. Among the isolates, Escherichia coli (51%) was the most prevalent, followed by Staphylococcus aureus (21%), Salmonella typhi (8.6%), Pseudomonas spp. (7.2%), Acinetobacter spp. (5.3%), Streptococcus spp. (4.9%), Klebsiella spp. (1.6%) and Neisseria gonorrhoeae (0.2%). Male patients (n = 94, 19%) were found to be infected mostly with Salmonella typhi (27%) whereas female patients (n = 395, 81%) were more susceptible to E. coli (58%). Among the isolates, 83% were MDR with an MAR index of 0.03-0.45. Overall, this study provides valuable insights into the current status of antibiotic resistance and helps develop strategies for effective management and containment of MDR infections.

Keywords: Multidrug resistance, MAR index, E. coli, Staphylococcus aureus, Salmonella typhi, Klebsiella spp., Pseudomonas spp.

Dedication

This thesis is dedicated to my parents, Md. Ahmadul Haque and Lubna Akther, for their unwavering support and love. Their confidence in me only reinforced the faint act of will I possessed to finish this project.

I then want to thank my Supervisor Dr. Muhammad Asaduzzaman, his guidance, encouragement, and expertise played a crucial role in the successful execution of this project.

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May the Divine bestow blessings on everybody who assisted me in my undertaking.

Thank you.

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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
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 history and evolution of antibiotics are closely intricately linked to the development process of modern medicine. Antibiotics are compounds produced by certain microorganisms, including bacteria and fungi, that can inhibit the growth of or kill other microorganisms . Infections caused by viruses and bacteria have always been a major problem for humans, however, before the development of antibiotics, many people were very susceptible to infection, while diseases such as tuberculosis (TB), meningitis and pneumonia either could not be treated or had only limited treatment options due to the emergence of drug-resistant pathogens [1].This vulnerability made the recurrence of massive pandemics an inevitable destiny for all human beings.

The era of antibiotics started with the discovery of penicillin in 1929 by Sir Alexander Fleming, a Scottish pharmacologist. The discovery of penicillin ushered in the era of antibiotics and Revolutionized treatment for bacterial infections since then, drastically decreasing the burden on public health globally. Since then, several other antibiotics have been discovered and developed, each with its own targets and modes of action against particular bacterial strains. [2].

Antibiotics changed medical care and rapidly produced desired results. Our ability to control many of these diseases, which had once killed or disabled large segments of our population, has led in turn to a worldwide decrease in mortality rates from infectious disease. That fall is recognized as

a major driver on the overall rise in life expectancy right here over these times. Despite this extensive and frequent irrational use of antibiotics has led to the emergence antibiotic resistance which is now recognized as a global threat to public health. These resistant bugs, sometimes referred to as the ‘superbugs’ are those that mutate and develop ways to fight the multiple antibiotics, making traditional treatments useless which then can help spread these diseases far and wide [3]. The situation underscores the importance of judicious antibiotic use and continued efforts to research and develop novel antimicrobials.

Major types of antibiotics

Key front-line antibiotics are penicillins, cephalosporins, macrolides, tetracyclines, fluoroquinolones and sulfonamides. These classes of antibiotics are also among the most widely used medications for treating bacterial infections [4]. In clinical practices, the major classes of antibiotics that are used are shown in Table 1:

Antibiotic Class	Examples	Bacteriostatic/Bactericidal	Gram +/-	MOA	Broad/Narrow spectrum
Aminoglycosides	Gentamicin Streptomycin	Bactericidal	-	Inhibits protein synthesis30s	Broad: abdominal infections & bacteremia
Cephalosporins	Ceftriaxone Cefepime	Bactericidal	+,-	Inhibits cell wall	Broad: Skin, urinary and respiratory infection
Tetracyclines	Tetracycline Doxycycline	Bacteriostatic	+,-	Inhibits protein synthesis 30s	Broad: Lime disease, PID, STIs
Penicillin	Ampicillin Amoxicillin	Bactericidal	+	Inhibits cell wall synthesis	Narrow: ENT, Burns & eye infection
Sulfonamides	Sulfasalazine Sulfamethoxazole	Bacteriostatic	+,-	Inhibits folate synthesis	Broad: : ENT, Burns & eye infection
Fluoroquinolones	Ciprofloxacin Levofloxacin	Bacteriostatic	+,-	Inhibits DNA replication	Broad: Respiratory & urinary infection
Macrolides	Azithromycin Erythromycin	Bactericidal	+	Inhibits protein synthesis 50s	Broad: Pneumonia, sinus, ENT, STIs
Carbapenems	Meropenem Ertapenem	Bactericidal	+,-	Inhibits cell wall synthesis	Broad: Urinary, intra-abdominal infection
Lincosamides	Clindamycin	Bacteriostatic	+	Inhibits protein synthesis	Narrow: Skin, bone & lung infections
Glycopeptides	Vancomycin	Bactericidal	+	Inhibits cell wall synthesis	Narrow: MRSA, Skin, and endocarditis infection

Table 1: Antibiotic class, examples, bacteriostatic/bactericidal, Gram+/-

Mechanism of action and broad/narrow spectrum threats [4]

1.2 Mechanism of action

Antibiotics function via many strategies for dealing with bacterial infections. These processes encompass interfering with cell wall production, protein synthesis, nucleic acid metabolism, and bacterial metabolic pathways. Different types of antibiotics work to destroy different parts or functions within bacterial cells, which stops the bacteria from growing and /or kills the germs. Many antibiotics work by doing specific activities in bacterial cells and are a type of potential antibacterial agent. Some antibiotics interfere with the biosynthesis of bacterial cell walls by inactivating enzymes essential for peptidoglycan formation. Several agents work by binding to bacterial ribosomes and inhibiting protein synthesis, while others prevent enzymes needed for DNA replication or transcription from placing nucleotides in sequence. In addition, some antibiotics affect bacterial metabolism when they act by blocking essential metabolic pathways. All these processes collectively work together to enable antibiotics to suppress bacterial multiplication or induce cell death which helps cure pathogenic infections [5]

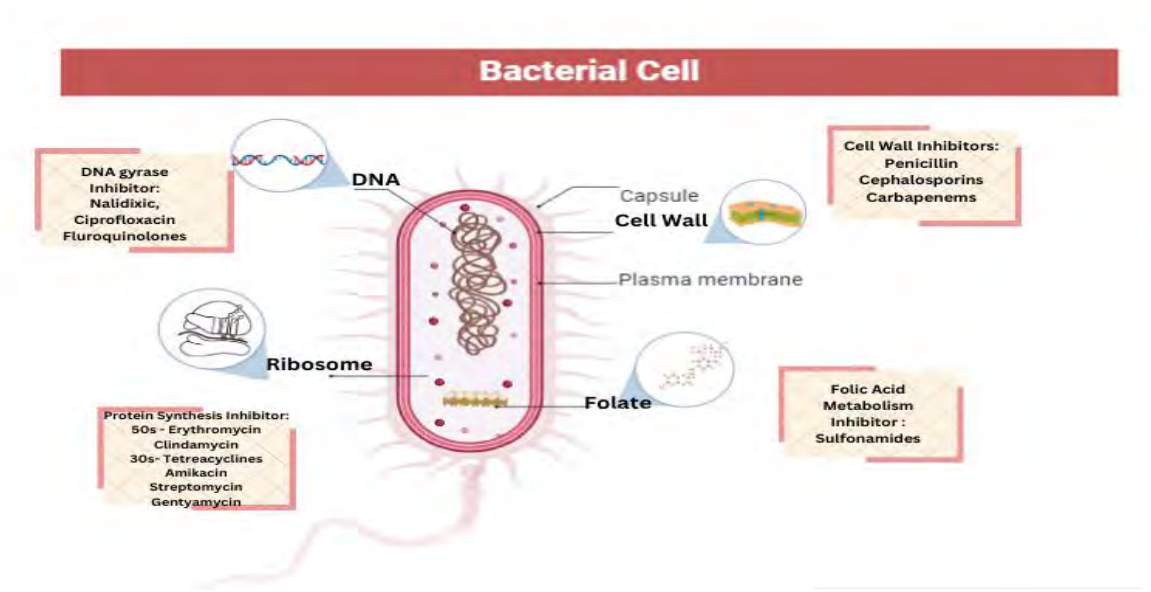


Figure 1: Mechanism of action of antibiotics.

1.3 Antibiotic resistance

Resistance to antibiotics is what happens when bacteria evolve and acquire the mechanisms to resist the therapeutic effects of antibiotic agents, making these treatments lose the efficacy of the drug to treat infections caused by certain types of bacterial reactions. This is mainly related to the abuse and misuse of antibiotics which puts a choice pressure on bacteria, leading to survival reproduction-resistant strains. In addition, the horizontal transfer of DNA between bacteria also allows resistance genes to spread. As a result, antibiotic-resistant infections pose greater and more extensive public health challenges, necessitating the development of alternative therapies and responsible good antibiotics use to reduce resistance.[6] .

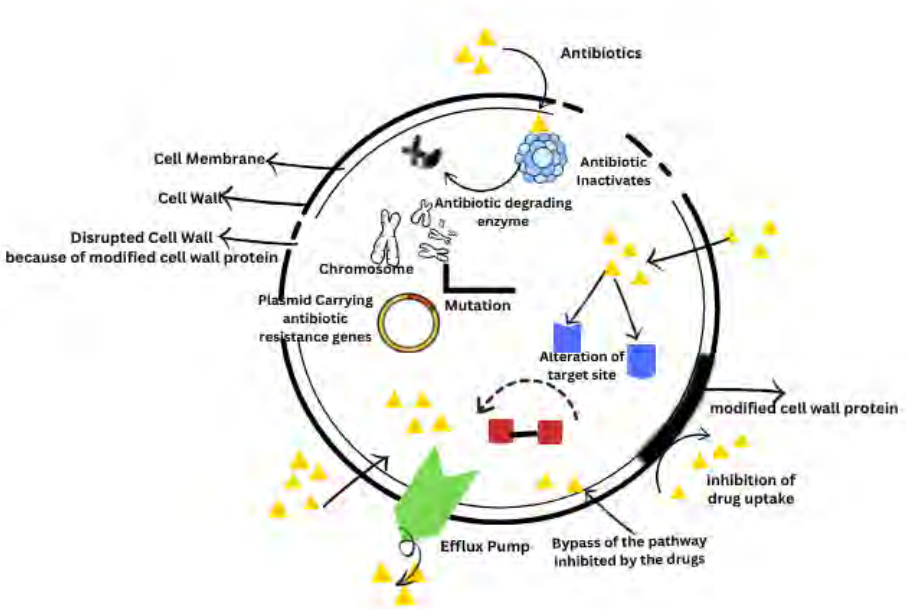


Figure 2: Antibiotic resistance mechanism.

1.4 Acquired resistance

The case of acquired resistance is when bacteria undergo genetic changes, such as mutations in their DNA or acquire genes from other organisms that enable them to survive the onslaught of antibiotics once effective against them. Similarly, resistance may also be facilitated by the horizontal transfer of genetic material (e.g., plasmids with such genes) among bacterial populations. This fact, rather than anything else that can be imagined addressing at the level of public policy or otherwise is why antibiotic stewardship began as an issue and this fundamentally reflects a problem in how we deal with bacterial infections. One of the major problems in dealing with bacterial infections comes from the fact that bacteria is able to evolve resistance due to every increasing number of methods they have at their disposal and so antibiotic usage needs to be restricted.

1.4.1 Genetic change

A genetic change is a mutation, in which the DNA sequence of an organism-changes due to addition or deletion apply. These changes can lead to the variability of traits and characteristics in an organism, which is important for evolution (generating genetic diversity within populations). In bacteria, mutations tend to be a very important mechanism promoting antibiotic resistance and adaptation to the environment (and hence survival success of bacterial communities). Methods of genetic change include various processes by which an alteration in the DNA sequence occurs such as mutation, genetic recombination, horizontal gene transfer (HGT), etc. The changes result in variations to the phenotype of an organism, which provides genetic diversity among populations and also affects evolutionary path. Within the bacterial world, changes in genetic material are instrumental for allowing adaptation to environmental adversities and usually leads to antibiotic

resistance phenomenon posing a role-defining effect on both the behavior of these populations and their ecological interactions with other surrounding microenvironments.

1.4.1 DNA transfer

The exchange of genetic material between organisms is known as DNA transfer. This is also referred to as DNA transfer from one organism to another. In bacteria, this can occur via conjugation, transformation and transduction among others as well by gene transfer in more complex organisms. The mechanisms make it possible for genetic information in the form of genes that control specific characteristics or functions to move from one organism among individuals and between populations. This is crucial for increasing genetic diversity and spreading gene functions like antibiotic resistance through the bacterial populations, affecting their ability to adapt and evolve.

1.4.3 Multi-drug resistance

Multidrug resistance, aka. MDR, describes the ability of microorganisms (such as bacteria and viruses) to resist the effects of more than one medication or drug. This is a significant challenge in treating infectious diseases because conventional medicines are not effective against these stubborn little microorganisms. Multidrug resistance needs multidisciplinary strategies to develop new treatments for effectively combating the emergence of resistant strains moving towards alarming proportions in healthcare. [7] .

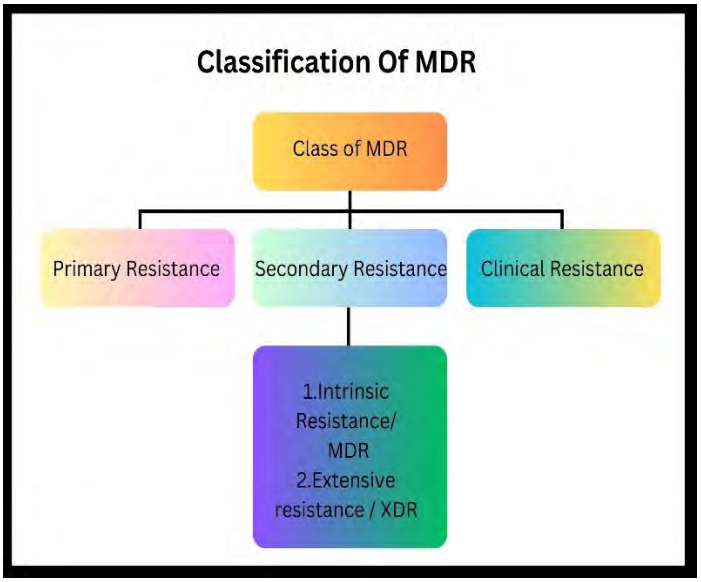


Figure 3: Classification of MDR. [7]

1.6.1 Mechanism of MDR

MDR may develop by a variety of mechanisms including the overexpression of drug efflux pumps, modifications in drug targets, and creation of de novo metabolic bypass routes. The acquisition of resistance genes by horizontal gene transfers also played an important role in the development of multidrug-resistant bacterial populations. These mechanisms act collectively to enable bacteria to fight against the effects of a wide range of antibiotics which leads to difficult clinical strategies and practices for dealing with bacterial infections. The mechanism of multidrug resistance amounts to the capabilities a bacterium has acquired to be resistant to more than one antibiotic through several different mechanisms. This includes genetic changes that lead to the overproduction of efflux pumps, pushing antibiotics outside the bacterial cell; altering antibiotic targets so drugs bind less strongly or not at all; and synthesis of enzymes removing antibiotics. [8]. In addition, bacteria can also receive horizontally transferred resistance genes that play an important role in the

emergence of multidrug resistance. These intricate processes enable bacteria can better survive the detrimental outcomes of a range of antibiotics, contributing significantly to problems with bacterial infection management and highlighting the importance for alternative treatment modalities as well as thoughtful antibiotic use. [9] .

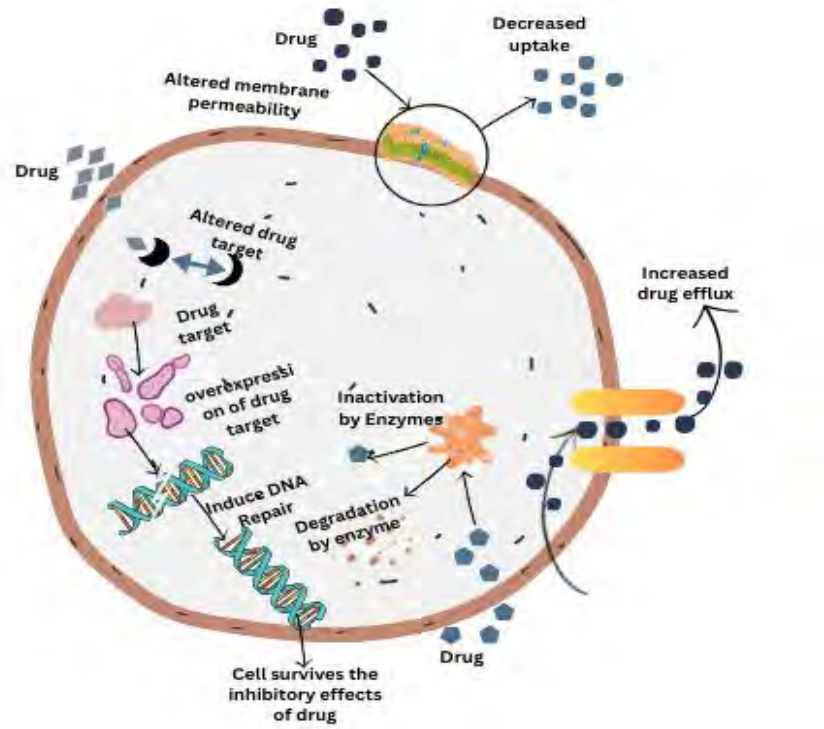


Figure 4 : Mechanism of multi-drug resistance.

1.7 Resistance mediated by efflux pumps

Efflux-based resistance occurs when bacteria develop the ability to export antibiotics out of their cells via specific efflux mechanisms. This shuts off antibiotic removal and allows it to accumulate, lowering its concentration within the bacterial cell and reducing how well it will work against the infection. Existing in many bacteria, this resistance mechanism is central to multidrug-resistant

infections, which makes it impossible for antibiotics of various types to reach the cells. Efflux pump-mediated resistance represents a serious issue in the treatment of bacterial infections, thereby reinforcing that there is an urgent requirement for novel strategies to target this type of drug resistance. [10] .

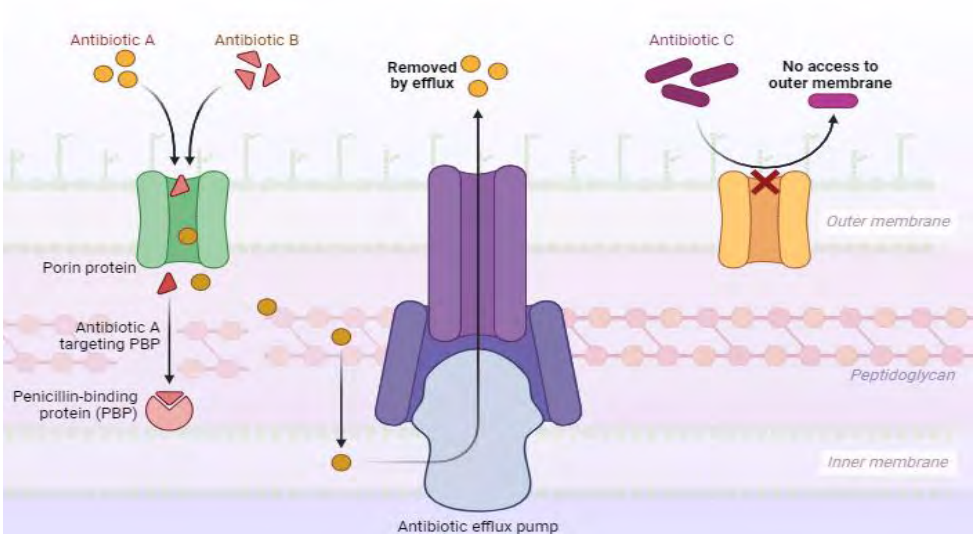


Figure 5: Multi-drug resistance facilitated by efflux pumps.

1.8 Resistance mediated by plasmid DNA

Resistance that is mediated by plasmids arises when bacteria obtain resistance genes that are housed on plasmids, which are small, circular DNA structures capable of being exchanged between bacterial cells. The presence of these resistance genes enables bacteria to endure the impact of antibiotics, playing a significant role in the emergence of antibiotic resistance within bacterial communities. The movement of plasmids that harbor resistance genes among bacteria enables the spread of resistance, creating considerable obstacles in the management of bacterial infections.

This mechanism of resistance underscores the necessity of tackling the dissemination of resistance genes located on plasmids and developing strategies to alleviate the effects of plasmid-mediated resistance in clinical environments. [11] .

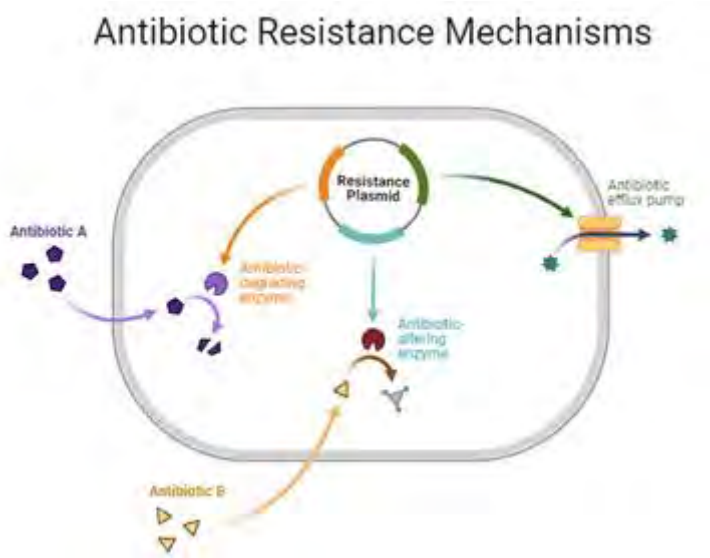


Figure 6: Antibiotic resistance mediated by plasmid DNA.

1.9 Resistance mediated by chromosomal DNA mutation

Resistance arising from mutations is when bacteria become resistant to the action of antibiotics through genetic changes in their chromosomal DNA. These mutations can result in the antibiotic's target site being altered, causing a reduction of affinity between them hence making it less effective for overall treatment. In addition, a mutation in chromosomal may lead to the over-expression of genes involved during resistance mechanisms against antibiotics. Mention resistance here, as the stems are often resistant to particular antibiotics and therefore can lead to bacteria becoming antibiotic-resistant. Understanding the role of chromosomal DNA mutations in antibiotic resistance is a necessary component for developing strategies for dealing with this aspect and

keeping antibiotics as useful tools in medicine. Mutation occurs through the following: 1. change in the target for antibacterial activities and 2. more efficient efflux systems, which pump out toxic molecules of the host. [12].

1.9.1 Target modification

Target modification refers to a process by which bacteria acquire resistance against antibiotics through modulating the target sites of these drugs. These changes could range from differences in the structure or assembly of target molecules (e.g. enzymes, cell wall components, etc.), to reduced binding affinity between antibiotics and their respective targets. The antibiotics then become less effective at stopping the bacteria from growing, or killing them. One of the most important modes is through target modification, driving home that bacteria are good at finding ways to evade antibiotics. Understanding the molecular aspects of target alteration is crucial to developing new strategies along that form novel ways for prevention and treatment against antibiotic resistance. [13].

1.9.2 Increased activation of efflux pumps

There are five classes of bacterial efflux pumps i. ATP-binding cassette(ABC) ii. major facilitator superfamily(MFS) iii. multidrug and toxic compound extrusion (MATE) iv. Resistance nodulation division(RND) v. small multidrug resistance family (SMR). Again there are some kinds of bacterial efflux pumps such as; i. multidrug efflux pumps ii.MexB iii. Tripartite efflux pumps iv. cmeABC pump v.mtrABC pump vi. single-component efflux systems. So The elevated activation of efflux pumps denotes the upregulation of these specialized transport proteins within bacterial cells, resulting in elevated levels of antibiotic expulsion from the cell. The increased activity of

efflux pumps leads to lower intracellular levels of antibiotics, thereby diminishing their efficacy in eliminating bacterial infections. The heightened activation of efflux pumps serves as a crucial mechanism of antibiotic resistance, allowing bacteria to eliminate antibiotics and persist in environments where these drugs are present. Grasping and tackling the elements that lead to the upregulation of efflux pumps is essential in fighting antibiotic resistance and formulating approaches to surmount this resistance mechanism. [14].

1.10 Multi-drug resistance pathogens

Multidrug resistance pathogens, aka MDR pathogens, are microorganisms that have acquired resistance to various classes of antibiotics (figure 7). The ability of these pathogens to endure the effects of various antimicrobial medications presents considerable obstacles in effectively treating infections caused by these resistant strains. The most notorious antibiotic-resistant bacteria can be summarized with the acronym ESKAPE, a collection of bacteria including – ***E****nterococcus faecium*, ***S****taphylococcus aureus*, ***K****lebsiella pneumoniae*, ***A****cinetobacter baumannii*, ***P****seudomonas aeruginosa*, and ***E****nterobacter spp* – that represent the primary contributors to hospital-acquired infections worldwide. ESKAPE pathogens are recovered preferentially from females than from males. [15].

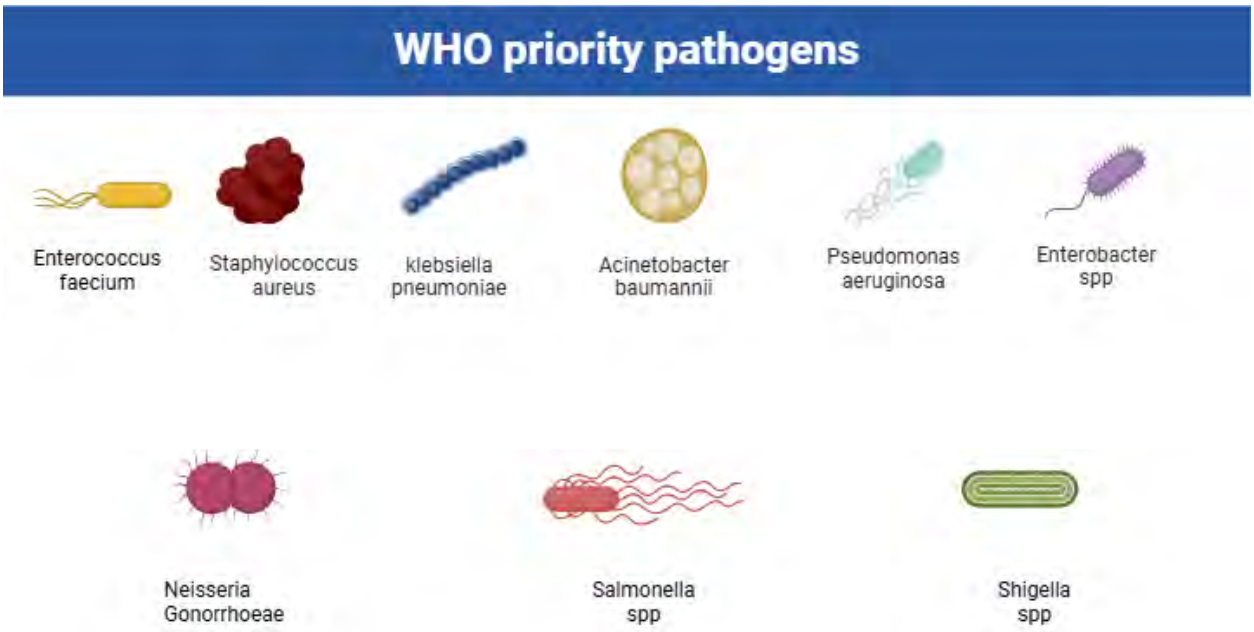


Figure 7: WHO priority pathogens. [16].

The pathogens currently included in GLASS-AMR are referred to as the GLASS pathogens and include: *Acinetobacter species*, *Escherichia coli*, *Klebsiella pneumoniae*, *Neisseria gonorrhoeae*, *Salmonella species*, *Shigella species*, *Staphylococcus aureus*, and *Streptococcus pneumonia*. These pathogens are also currently included in the priority list of the WHO for antibiotic surveillance.[16].

Chapter 2

Objective of the study

To understand the acquisition of emerging resistance to multiple drugs and its magnitude, this study was designed to investigate the prevalence and antimicrobial resistance pattern among the patients from selected hospitals of Dhaka, Bangladesh. An understanding of MDR by health professionals and researchers may lead to new approaches aimed at overcoming this resistance for more effective therapeutic interventions and better patient outcomes. Understanding MDR, moreover, might help in the design of new pharmaceuticals and additional therapeutic strategies to overcome or reduce antimicrobial resistance (AMR) processes thus guaranteeing continued efficacy in antibiotic therapy. Understanding both MDR and AMR is crucial for controlling and preventing a crisis from emergent resistant infections, as well as to improve healthcare practices in treatment against wide spreading disorders.

Chapter 3

Methodology

3.1 Study settings

A retrospective analysis was conducted utilizing 489 isolates from the Microbiology Laboratory of Delta Healthcare Mirpur Limited between February 2022 and March 2024.

3.2 Types of sample

Samples were collected retrospectively from various biological specimens including -

- Urine
- Blood
- Pus
- Sputum
- Stool
- Vaginal swab
- Throat swab
- Tracheal aspirates
- ET tube

3.3 Data collection

A predesigned questionnaire (Figure 3.1) was used for the collection of data including the following information:

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 8: 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 Source of data collection

Data on antimicrobial sensitivity patterns and other information were collected using the above questionnaire from the hospital database.

3.4 MAR index determination

The multiple antibiotic resistance (MAR) index is calculated by employing this formula. [17]

MAR Index = a / b

where,

a = total number of antibiotics to which an isolate was resistant

b = total number of antibiotics utilized in the assessment of antimicrobial susceptibility test.

3.5 Statistical analysis

The data comparing males and females were examined for statistical significance by calculating *p*-values using Microsoft Excel, 2018.

Chapter 4

Results & Discussion

4.1. Culture positivity of specimen

Culture and susceptibility testing were performed on clinical specimens that fulfilled the American Society for Microbiology (ASM) standards. Specimens were swiftly sent to the microbiological lab for additional processing upon arrival at the collecting sites. Positive growth was seen in 489 out of 1890 processed specimens (25.87%). Figure 9 shows that pathogen-induced infection was present among the 489 positive samples evaluated for infection. Isolates of various bacteria were found in the samples. The most prevalent was *Escherichia coli* (n = 250, 51.12%), followed by *Staphylococcus aureus* (n = 103, 21.01%), *Klebsiella spp.* (n = 8, 1.63%), *Salmonella typhi* (n = 42, 8.58%), *Acinetobacter spp.* (n = 26, 5.31%), *Pseudomonas spp.* (35, 7.15%) *Streptococcus spp.*(n = 24, 4.90%) and *Neisseria gonorrheae* (n = 1, 0.20%).

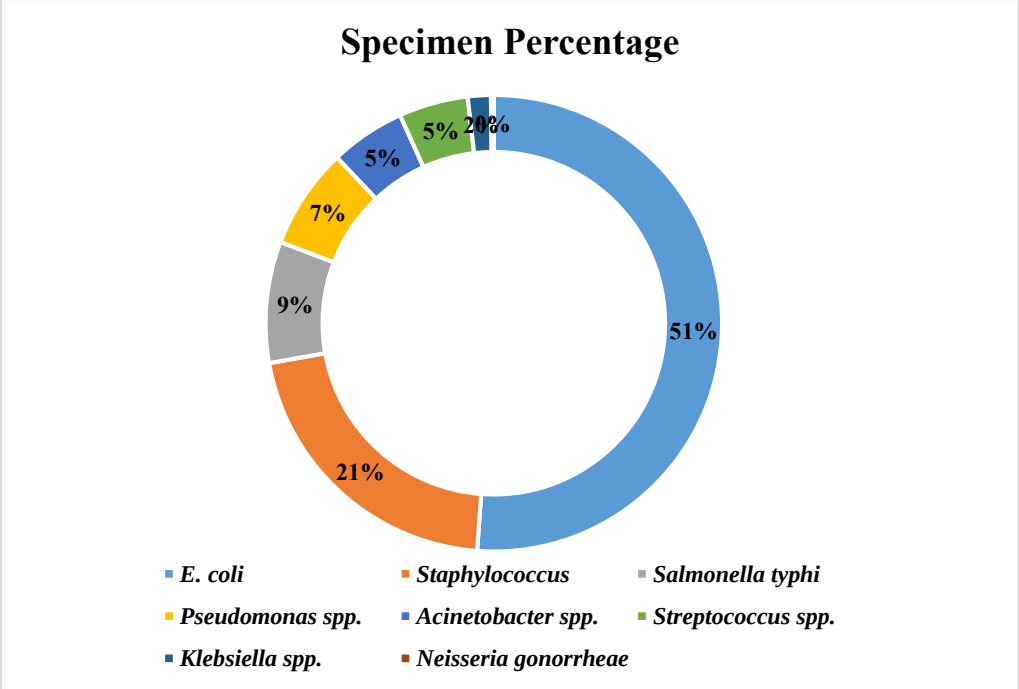


Figure 9: Percentage of pathogens.

4.2 Pathogens distribution in males and females

Among the pathogens, *E. coli* was more common in female patients (57.4%) than in males (24.4%). On the other hand, *Salmonella typhi* was more prevalent in males (26.5) than females (4.3). We can understand it better by the following Table 2.

Pathogens Name	**No. of male**	**%**	**No. of female**	**%**
E. Coli	23	**24.5**	227	**57.5**
Klebsiella spp.	2	**2.1**	6	**1.5**
Pseudomonas spp.	6	**6.4**	29	**7.3**
Salmonella typhi	25	**26.6**	17	**4.3**
Streptococcus spp.	10	**10.6**	14	**3.5**
Acinetobacter spp.	4	**4.3**	22	**5.6**
Staphylococcus spp.	23	**24.5**	80	**20.3**
Neisseria gonorrhoeae	1	**1.0**	0	**0**
TOTAL	**94**		**395**	

Table 2. Pathogen distribution in males and females

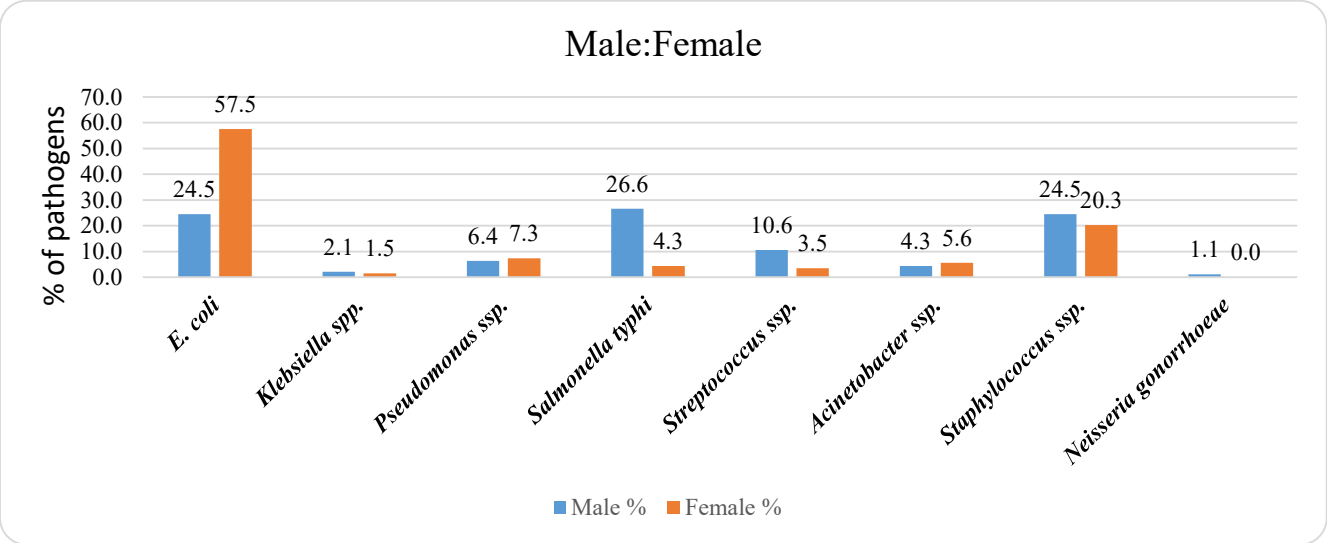


Figure 10: Percentage of pathogen among male and female.

4.3 Distribution of Pathogens Among the Diverse Clinical Specimens

In the analysis of the clinical specimens, the highest frequency of pathogen isolation occurred in urine specimens, followed by blood, pus, sputum, stools, wound, throat and vaginal swabs, tracheal aspirates, and ET tubes. (Table 3)

Specimen Type	**No.**	**Percentage (%)**
Urine	366	74.8
Blood	69	14.1
Sputum	2	0.40
Pus	20	4.08
Stool	7	1.43
Wound swab	4	0.81
Throat swab	6	1.22
Tracheal aspirate	1	0.20
Et tube	1	0.20
Vaginal swab	12	2.45
Urethra	1	0.20
Total	**489**	**100**

Table 3. Distribution of pathogens among the diverse clinical specimens.

4.4 Specimen Found in Male and Female

Table 4 shows the distribution of samples among the males vs. females.

Specimen type	Male		Female	
	No.	%	No.	%
Blood	43	45.7	26	6.6
Urine	33	35.1	333	84.3
Sputum	1	1.1	1	0.3
Pus	7	7.4	13	3.3
Stool	3	3.2	4	1.0
Wound swab	1	1.1	3	0.8
Throat swab	5	5.3	1	0.3
Tracheal aspirate	0	0.0	1	0.3
urethra	1	1.1	0	0.0
Vaginal Swab	0	0.0	12	3.0
ET tube	0	0.0	1	0.3
Total	**94**	100.0	**395**	100.0

Table 4: Types of specimens of male and female

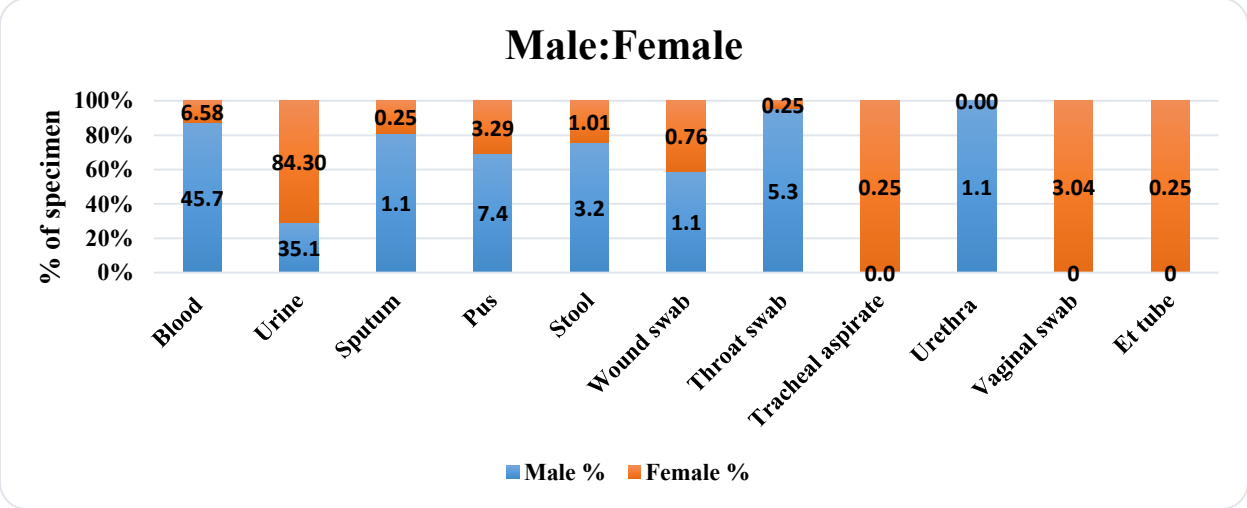


Figure 11: Specimen types, number, and percentage of males vs. females.

4.5 Age group of patients infected by pathogens.

The highest number of patients was observed in the age group of 1 to 10 year, followed by the 11 to 20 year age group for males, while for females, the most significant number was also in the 1 to 10 year age group, followed by the 21 to 30 year age group, (Table 5, Figure 12).

Age Range (year)	No. of Male	Percentage (%)	No. of Female	Percentage (%)
0-1	11	12	14	3
1-10	26	28	30	8
11-20	15	16	16	4
21-30	12	13	143	36
31-40	4	4	90	23
41-50	5	5	32	8
51-60	3	3	38	10
61-70	6	7	18	4
71-80	5	5	11	3
81-90	3	3	3	1
91-100	4	4	0	0

Table 5. Age range of male & female patients infected by pathogens

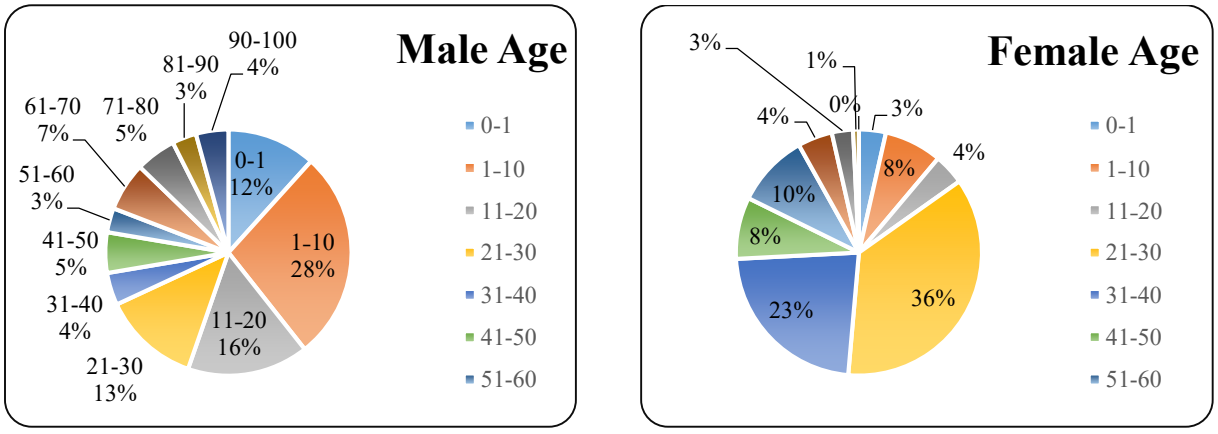


Figure 12: Age range of male vs female with percentage

4.6 Antibiotic susceptibility pattern of pathogens

4.6.1 Antibiotic susceptibility pattern of *Escherichia coli*

A substantial percentage of *E. coli* isolates (80.4%) exhibited resistance to cefixime, followed by cefotaxime (79%) and gentamicin (61.5%) (Table 6). On the other hand, azithromycin demonstrated the highest sensitivity (85.9%), followed by sulfomethoxazole (82.5%) and imipenem (81.5%).

Antibiotic	Sensitive	Sensitive(%)	Resistant	Resistant(%)
AMK (Amikacin)	67	78.82	18	21.17
AMC (Amoxiclav)	62	63.2	36	36.7
AMX (Amoxicillin)	12	27.2	32	72.7
ATM(Aztreonam)	8	80.0	2	20.0
CEF(Cefepime)	148	63.7	84	36.2
CFM (Cefixime)	48	19.5	198	80.4
CAZ (Ceftazidime)	52	50.4	51	49.5
CXM (Cefuroxime)	20	62.5	12	37.5
CE (Cephradine)	30	14.9	171	85.0
C (Chloramphenicol)	24	75.0	8	25.0
CIP (Ciprofloxacin)	162	60.6	105	39.3
GM (Gentamicin)	98	38.5	156	61.5
MEM (Meropenem)	35	79.5	9	19.5
NET (Netilimicin)	12	80.0	3	20.0
VA (Vancomycin)	1	12.5	7	87.5
SMX (Sulfamethoxazole)	139	82.5	27	17.5
LZD (Linezolid)	0	0	10	100
AZM (Azithromycin)	92	85.9	15	14.1
CT (Colistin)	6	31.5	13	69.5
NAL (Nalidixic acid)	34	77.2	10	22.8
IPM (Imipenem)	40	81.6	9	19.4
CTX (Cefotaxime)	52	21.0	195	79.0
LFX (Levofloxacin)	61	31.9	130	68.1
SXT (Cotrimoxazole)	1	0.0	1	0.0
NIT (Nitrofurantoin)	1	25.0	3	75.0
CRO (Ceftriaxone)	41	50.6	40	49.4
DO (Doxycycline)	1	100	0	0
ERY (Erythromycin)	1	0	1	0
TE (Tetracycline)	73	64.6	40	35.4

Table 6: Antibiotic susceptibility pattern for Escherichia coli.

4.6.2 Antibiotic susceptibility pattern of *Staphylococcus aureus*

A substantial percentage of *S. aureus* isolates (96%) exhibited sensitive to amoxiclav, followed by cefuroxime (92.8%) (Table 7). Aztreonam demonstrated the highest resistance (90%), followed by azithromycin (83.7%).

Antibiotics	Sensitive	Sensitive (%)	Resistant	Resistant (%)
AMK (Amikaicin)	42	55.2	34	44.8
AMC (Amoxiclav)	24	96.0	1	4.0
AMX (Amoxicillin)	6	60.0	4	40.0
ATM(Aztreonam)	1	10.0	9	90.0
CEF(Cefepime)	35	42.6	47	57.4
CFM (Cefixime)	24	33.8	47	66.1
CAZ (Ceftazidime)	10	37.0	17	63.0
CXM (Cefuroxime)	13	92.8	1	7.2
CE (Cephradine)	17	27.4	45	72.4
C (Cholramphenicol)	7	87.5	1	12.5
CIP (Ciprofloxacin)	50	60.2	33	39.8
GM (Gentamicin)	13	30.2	30	69.8
MEM (Meropenem)	35	79.5	9	20.5
NET (Netilimicin)	2	0	0.0	0
VA (Vancomycin)	48	81.3	11	18.7
SMX (Sulphomethoxazole)	48	64	27	36
LZD (Linezolid)	23	76.6	7	23.4
AZM (Azithromycin)	13	16.2	67	83.7
CT (Colistin)	6	31.5	13	68.5
NAL (Nalidixic acid)	2	22.2	7	77.8
IPM (Imipenem)	10	83.3	2	16.7
CTX (Cefotaxime)	20	74.0	7	26.0
LFX (Levofloxacin)	61	82.4	13	17.6
SXT (Cotrimoxazole)	0.0	0.0	2.1	0.0
NIT (Nitrofurantoin)	1	25	3	75
CRO (Ceftriaxone)	41	50.6	40	49.3
DO (Doxycycline)	7	77.7	2	22.3
ERY (Erythromycin)	0	0	9	0
TE (Tetracycline)	13	26	37	74

Table 7: Antibiotic susceptibility pattern for Staphylococcus aureus

4.6.3 Antibiotic susceptibility pattern of *Salmonella typhi*

A substantial percentage of *Salmonella typhi* isolates (94.1%) exhibited highest sensitivity to meropenem, followed by ceftazidime (92.3%) and cholramphenicol (90.9%) (Table 8). Nalidixic acid demonstrated the highest sensitivity of 83.4%, followed by cephradine (75.0%) and gentamicin (72.3%).

Antibiotics	Sensitive	Sensitive (%)	Resistant	Resistant (%)
AMK (Amikaicin)	30	88.2	4	11.8
AMC (Amoxiclav)	9	81.8	2	18.2
AMX (Amoxicillin)	9	69.2	4	30.8
AMP (Ampicillin)	2	0	0	0
ATM(Aztreonam)	3	75	1	25
CEF(Cefepime)	37	90.2	4	9.8
CFM (Cefixime)	21	84.0	4	16
CAZ (Ceftazidime)	24	92.3	2	7.7
CXM (Cefuroxime)	1	0	1	0
CE (Cephradine)	5	25.0	15	75.0
C (Cholramphenicol)	10	90.9	1	9.1
CIP (Ciprofloxacin)	18	69.2	8	30.7
GM (Gentamicin)	5	27.7	13	72.3
MEM (Meropenem)	16	94.1	1	5.9
NET (Netilimicin)	4	0	0.0	0
VA (Vancomycin)	2	40	3	60
SMX (Sulphomethoxazole)	31	81.5	7	18.5
LZD (Linezolid)	0	0	0.0	0
AZM (Azithromycin)	0	0	7	0
DA (Clindamycin)	0.0	0.0	0.0	0.0
CT (Colistin)	3	0	3	0
CFX (Cefoxitin)	0.0	0.0	0.0	0.0
NAL (Nalidixic acid)	1	16.6	5	83.4
IPM (Imipenem)	5	83.4	1	16.6
CTX (Cefotaxime)	10	90.9	1	9.1
LFX (Levofloxacin)	24	82.7	5	17.3
SXT (Cotrimoxazole)	0.0	0.0	2.1	0.0
NIT (Nitrofurantoin)	13	0	13	0
CRO (Ceftriaxone)	35	89.7	4	10.3
DO (Doxycycline)	0	0	0	0
TE (Tetracycline)	13	86.6	2	13.4

Table 8: Antibiotic susceptibility pattern for Salmonella typhi

4.6.4 Antibiotic susceptibility pattern of *Klebsiella spp.*

A substantial percentage of *Klebsiella spp.* isolates exhibited resistance to nitrofurantoin (75%), followed by cephradine (66.7%) (Table 9). Cefepime along with ciprofloxacin demonstrated the highest sensitivity (85.7%), followed by cefixime (85.0%).

Antibiotics	Sensitive	Sensitive (%)	Resistant	Resistant (%)
AMK (Amikaicin)	4	80.0	1	20.0
AMC (Amoxiclav)	4	80.0	1	20.0
AMX (Amoxicillin)	1	0	1	0
AMP (Ampicillin)	0	0	0	0
ATM(Aztreonam)	0	0	0.0	0.0
CEF(Cefepime)	6	85.7	1	14.3
CFM (Cefixime)	6	85.0	2	14.0
CAZ (Ceftazidime)	5	71.4	2	28.6
CE (Cephradine)	1	33.3	2	66.7
C (Cholramphenicol)	0	0	0	0
CIP (Ciprofloxacin)	6	85.7	1	14.3
MEM (Meropenem)	6	0	0	0
NET (Netilimicin)	0	0	0.0	0
VA (Vancomycin)	0	0	0	0
SMX (Sulphomethoxazole)	2	0	2	0
Cloxacillin	0	0.0	0.0	0.0
LZD (Linezolid)	0	0	0.0	0
AZM (Azithromycin)	0	0	7	0
DA (Clindamycin)	0.0	0.0	0.0	0.0
CT (Colistin)	0	0	2	0
CFX (Cefoxitin)	0.0	0.0	0.0	0.0
NAL (Nalidixic acid)	0	0	0	0
IPM (Imipenem)	0	0	0	0
CTX (Cefotaxime)	3	0	0	0
LFX (Levofloxacin)	4	80.0	1	20.0
SXT (Cotrimoxazole)	0.0	0.0	2.1	0.0
NIT (Nitrofurantoin)	1	25.0	3	75.0
CRO (Ceftriaxone)	6	75.0	2	25.0
DO (Doxycycline)	0	0	0	0
ERY (Erythromycin)	0	0	0	0
PRL (Piperacillin)	0.0	0.0	0.0	0.0
TE (Tetracycline)	2	0	0.0	0.0

Table 9: Antibiotic susceptibility pattern for Klebsiella spp.

4.6.5 Antibiotic susceptibility pattern of *Acinetobacter spp.*

A substantial percentage of *Acinetobacter spp.* isolates (75.0%) exhibited resistance to cephradine, followed by cefixime (73.3%) (Table 10). *Acinetobacter spp.* demonstrated the highest sensitivity against meropenem (88.8%), followed by levofloxacin (87.5%) and nitrofurantoin (78.5%).

Antibiotics	Sensitive	Sensitive (%)	Resistant	Resistant (%)
AMK (Amikacin)	13	76.4	4	23.5
AMC (Amoxiclav)	0	0	7	0
AMX (Amoxicillin)	4	0	4	0
AMP (Ampicillin)	0	0	0	0
ATM (Aztreonam)	0	0	0	0.0
CEF (Cefepime)	18	75.0	6	25.0
CFM (Cefixime)	4	26.6	11	73.3
CAZ (Ceftazidime)	3	0	0	0
CXM (Cefuroxime)	1	33.3	2	66.6
CE (Cephradine)	4	25.0	12	75.0
C (Chloramphenicol)	4	0	0	0
CIP (Ciprofloxacin)	8	66.6	4	33.3
GM (Gentamicin)	7	0	7	0
MEM (Meropenem)	8	88.8	1	11.1
NET (Netilimicin)	4	0	0.0	0
VA (Vancomycin)	5	0	0	0
SMX (Sulfamethoxazole)	10	71.4	4	28.5
LZD (Linezolid)	0	0	2	0
AZM (Azithromycin)	6	28.5	15	71.4
CT (Colistin)	5	38.4	8	61.5
NAL (Nalidixic acid)	0	0	4	0
IPM (Imipenem)	7	0	0	0
CTX (Cefotaxime)	8	72.7	3	27.2
LFX (Levofloxacin)	21	87.5	3	12.5
SXT (Cotrimoxazole)	0.0	0.0	1	0.0
NIT (Nitrofurantoin)	11	78.5	3	21.4
CRO (Ceftriaxone)	14	66.6	7	33.3
DO (Doxycycline)	3	37.5	5	62..5
ERY (Erythromycin)	2	28.5	5	71.4
TE (Tetracycline)	5	62.5	3	37.5

Table 10: Antibiotic susceptibility pattern for Acinetobacter spp.

4.6.6 Antibiotic susceptibility pattern of *Pseudomonas spp.*

A substantial percentage of *Pseudomonas spp.* isolates (90.9%) exhibited sensitivity to meropenem, followed by amikacin (81.4%). Cephradine demonstrated the highest resistance (92.3%), followed by azithromycin (90.9%).

Antibiotics	Sensitive	Sensitive (%)	Resistant	Resistant (%)
AMK (Amikaicin)	22	81.4	5	18.6
AMC(Amoxiclav)	8	53.3	7	46.7
AMX (Amoxicillin)	0	0	3	0
AMP (Ampicillin)	0	0	0	0
ATM(Aztreonam)	0	0	0	0.0
CEF(Cefepime)	16	45.7	19	54.3
CFM (Cefixime)	4	18.1	18	81.9
CAZ (Ceftazidime)	9	37.5	15	62.5
CXM (Cefuroxime)	0	0	1	0
CE (Cephradine)	1	7.7	12	92.3
C (Chloramphenicol)	3	60.0	2	40.0
CIP (Ciprofloxacin)	20	76.9	6	23.1
GM (Gentamicin)	5	0	13	0
MEM (Meropenem)	10	90.9	1	9.1
NET (Netilimicin)	0	0	0.0	0
VA (Vancomycin)	0	0	0	0
SMX (Sulfamethoxazole)	7	24.2	22	75.8
LZD (Linezolid)	1	0	0.0	0
AZM (Azithromycin)	3	9.1	30	90.9
CT (Colistin)	3	0	3	0
NAL (Nalidixic acid)	0	0	11	0
IPM (Imipenem)	4	80.0	1	20.0
CTX (Cefotaxime)	1	0	0	0
LFX (Levofloxacin)	25	78.1	7	21.9
SXT (Cotrimoxazole)	0.0	0.0	2.1	0.0
NIT (Nitrofurantoin)	15	45.4	18	54.6
CRO (Ceftriaxone)	7	21.8	25	78.2
DO (Doxycycline)	0	0	0	0
ERY (Erythromycin)	0	0	0	0
TE (Tetracycline)	6	40.0	9	60.0

Table 11: Antibiotic susceptibility pattern for pseudomonas spp.

4.6.7 Antibiotic susceptibility pattern of *Neisseria gonorrheae*

Neisseria Gonorrheae exhibited resistance to cefixime, followed by ceftazidime and cephradine (Table 12). Amikacin demonstrated sensitivity in some antibiotics like amoxiclave, cefepime, ciprofloxacin .

Antibiotics	Sensitive	Sensitive (%)	Resistant	Resistant (%)
AMK (Amikaicin)	-	0	-	0
AMC (Amoxiclave)	1	0	0	0
AMX (Amoxicillin)	-	0	-	0
AMP (Ampicillin)	-	0	-	0
ATM(Aztreonam)	-	0	-	0
CEF(Cefepime)	1	0	0	0
CFM (Cefixime)	0	0	1	0
CAZ (Ceftazidime)	0	0	1	0
CXM (Cefuroxime)	-	0	-	0
CE (Cephradine)	-	0	1	0
C (Chloramphenicol)	1	0	0	0
CIP (Ciprofloxacin)	1	0	0	0
GM (Gentamicin)	1	0	0	0
MEM (Meropenem)	1	0	0	0
NET (Netilimicin)	-	0	-	0
SMX (Sulfamethoxazole)	-	0	-	0
LZD (Linezolid)	-	0	-	0
AZM (Azithromycin)	-	0	-	0
CT (Colistin)	-	0	-	0
NAL (Nalidixic acid)	-	0	-	0
IPM (Imipenem)	-	0	-	0
CTX (Cefotaxime)	1	0	0	0
LFX (Levofloxacin)	-	0	-	0
SXT (Cotrimoxazole)	-	0	-	0
NIT (Nitrofurantoin)	1	0	0	0
CRO (Ceftriaxone)	1	0	0	0
DO (Doxycycline)	-	0	-	0
ERY (Erythromycin)	-	0	-	0
TE (Tetracycline)	-	0	-	0

Table 12: Antibiotic susceptibility pattern for Neisseria gonorrheae.

4.6.8 Antibiotic susceptibility pattern of *Streptococcus spp.*

A substantial percentage of *Streptococcus* isolates (93.3%) exhibited resistance to cefixime, followed by aztreonam (80.0%) and colistin (75.0%) (Table 13). Nitrofurantoin showing the highest sensitivity that's (89.4%), followed by meropenem (88.8%), and amikacin (87.5%).

Antibiotics	Sensitive	Sensitive(%)	Resistant	Resistant(%)
AMK (Amikaicin)	21	87.5	1	12.5
AMC (Amoxiclave)	8	80.0	2	20.0
AMX (Amoxicillin)	0	0	1	0
AMP (Ampicillin)	0	0	0	0
ATM(Aztreonam)	1	20.0	4	80.0
CEF(Cefepime)	12	54.5	10	45.5
CFM (Cefixime)	1	6.6	14	93.3
CAZ (Ceftazidime)	3	37.5	5	62.5
CXM (Cefuroxime)	3	0	0	0
CE (Cephradine)	4	28.5	10	71.5
C (Chloramphenicol)	1	0	0	0
CIP (Ciprofloxacin)	12	60. 0	8	40.0
GM (Gentamicin)	3	30.0	7	70.0
MEM (Meropenem)	8	88.8	1	11.2
NET (Netilimicin)	0	0	0.0	0
VA (Vancomycin)	10	71.5	4	28.5
SMX(Sulfamethoxazole)	4	28.5	10	71.5
LZD (Linezolid)	12	80.0	3	20.0
AZM (Azithromycin)	6	28.5	15	74.5
CT (Colistin)	1	25.0	3	75.0
NAL (Nalidixic acid)	0	0	1	0
IPM (Imipenem)	0	0	2	0
CTX (Cefotaxime)	2	40.0	3	60.0
LFX (Levofloxacin)	7	77.7	2	22.3
SXT (Cotrimoxazole)	0.0	0.0	2.1	0.0
NIT (Nitrofurantoin)	17	89.4	2	10.6
CRO (Ceftriaxone)	9	0	9	0
DO (Doxycycline)	0	0	0	0
ERY (Erythromycin)	0	0	0	0
TE (Tetracycline)	4	40.0	6	60.0

Table 13: Antibiotic susceptibility pattern for Streptococcus ssp.

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

According to Table 14, amikacin shows greater sensitivity in both males and females. On the other hand, azithromycin shows greater resistance in both male and female (Table 14).

Antibiotic	Male Sensitive (%)	Female Sensitive (%)	Male Resistant (%)	Female Resistant (%)
AMK (Amikacin)	62.8	60.8	17.0	11.1
AMC (Amoxiclave)	25.5	25.3	8.5	9.6
AMX (Amoxicillin)	10.6	5.6	7.4	10.6
AMP (Ampicillin)	8.5	1.3	3.2	3.3
ATM(Aztreonam)	1.1	0.3	0.0	0.0
CEF(Cefepime)	64.9	24.8	23.4	36.7
CFM (Cefixime)	23.4	21.3	40.4	48.4
CAZ (Ceftazidime)	34.0	23.0	10.6	19.0
CXM (Cefuroxime)	1.1	4.8	4.3	3.0
CE (Cephradine)	18.1	42.3	46.8	8.1
C (Chloramphenicol)	6.4	10.9	4.3	2.0
CIP (Ciprofloxacin)	44.7	42.5	27.7	24.3
GM (Gentamicin)	21.3	26.6	25.5	27.3
MEM (Meropenem)	43.6	40.0	7.4	2.8
NET (Netilimicin)	5.3	4.1	0.0	1.0
VA (Vancomycin)	21.3	11.6	2.1	5.3
SMX (Sulphomethoxazole)	38.3	42.0	33.0	33.4
LZD (Linezolid)	12.8	6.3	0.0	5.6
AZM (Azithromycin)	14.9	10.89	61.7	77.70
CT (Colistin)	6.4	16.7	10.6	17.0
NAL (Nalidixic acid)	2.1	3.3	10.6	14.9
IPM (Imipenem)	9.6	13.7	1.1	4.6
CTX (Cefotaxime)	25.5	12.9	9.6	10.1
LFX (Levofloxacin)	53.2	48.1	14.9	25.8
SXT (Cotrimoxazole)	0.0	0.0	2.1	0.0
NIT (Nitrofurantoin)	42.6	15.7	18.1	59.2
CRO (Ceftriaxone)	53.2	32.9	28.7	52.9
DO (Doxycycline)	3.2	2.0	1.1	1.5
ERY (Erythromycin)	1.1	0.8	3.2	3.0
TE (Tetracycline)	23.4	15.4	20.2	27.1

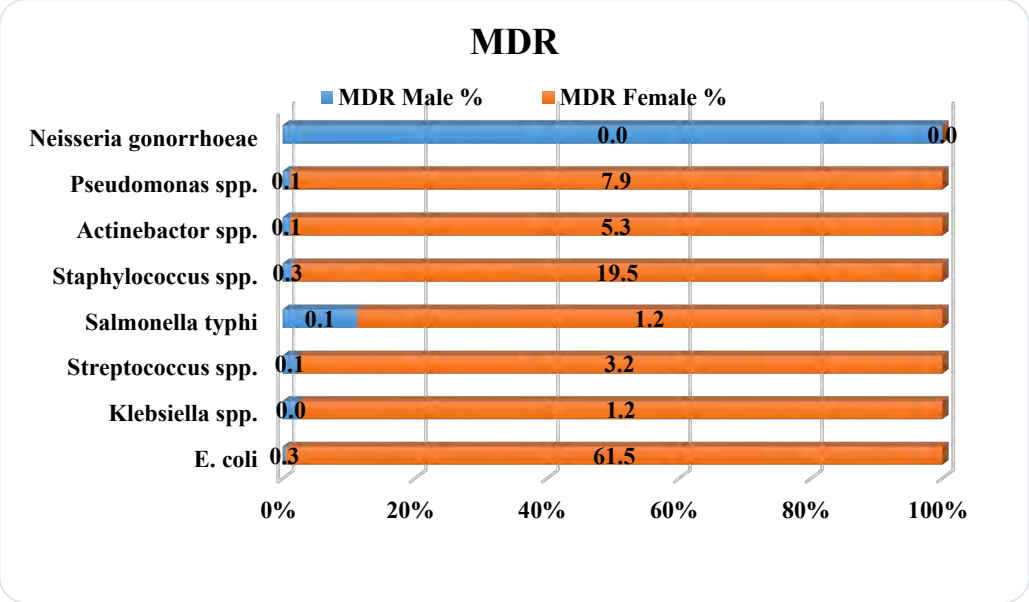
Table 14: Comparison of different Antibiotic sensitivity and resistant pattern for male and female

4.8 Multi-drug resistance and non- multi drug resistance

(Table 15) shows that compared to male patients (0.30%); female patients had a higher proportion of MDR *E. coli* (61.50%). The same goes for the other pathogens females have a higher rate of MDR than men. Additionally, just 3 male patients had non-MDR *E. coli* activity compared to 19 female cases .

Name of Pathogens	**MDR**				**Non-MDR**			
	Male		**Female**		**Male**		**Female**	
	No.	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
E.Coli	20	0.30	208	61.50	3	11.1	19	33.3
Klebsiella spp.	2	0.03	4	1.19	0	0	2	3.5
Streptococcus spp.	5	0.07	11	3.20	5	18.5	3	5.3
Salmonella Typhi	10	0.15	4	1.18	15	55.6	13	22.8
Staphylococcus spp.	19	0.28	66	19.52	4	14.8	14	24.6
Actinebactor spp.	4	0.06	18	5.32	0	0.0	4	7.0
Pseudomonus spp.	6	0.09	27	7.98	0	0.0	2	3.5
Neisseria gonorrhoeae	1	0.01	0	0.00	0	0	0	0
Total	**67**		**338**		**27**		**57**	

Table 15: Percentage of MDR and Non-MDR pathogens among male and female



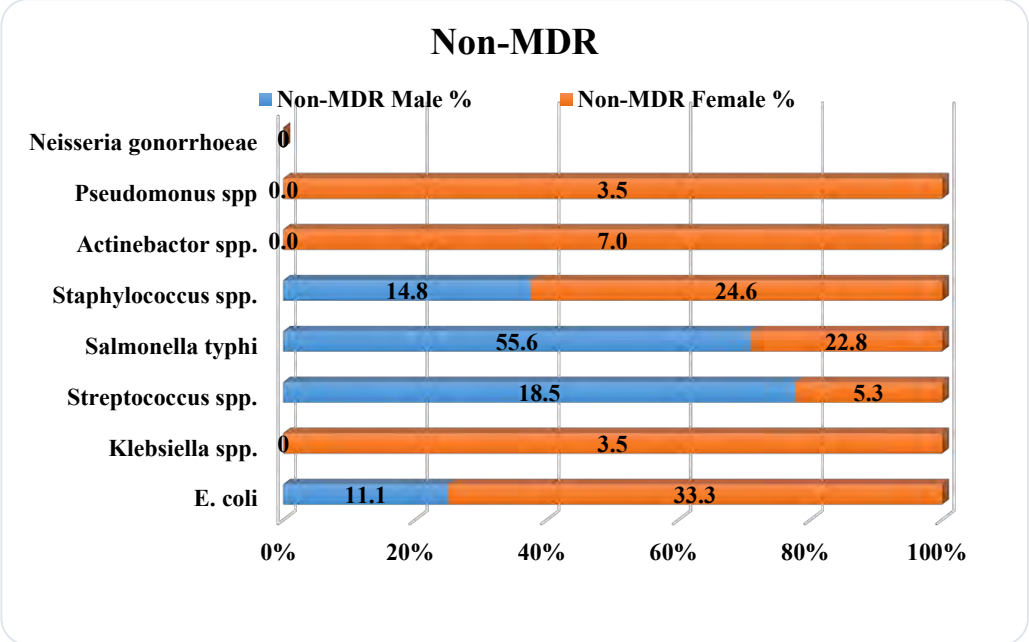


Figure 13: Percentage of distribution of MDR & Non-MDR pathogens between male and female

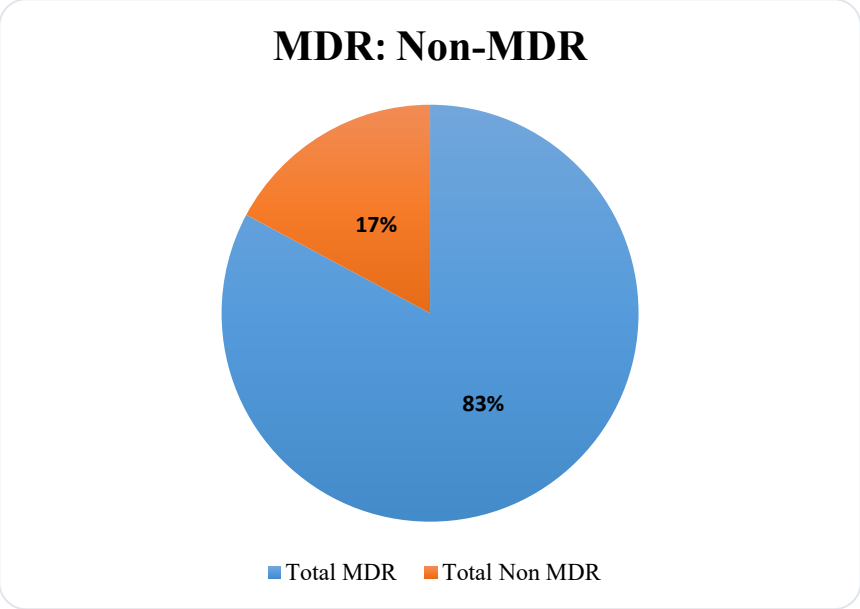


Figure 14: Ratio of MDR and non-MDR bacteria

Looking at the figure 14, it is clear that 83% of total sample shows multidrug resistance and the rest 17% is Non-MDR . Which means the amount of MDR is very high in this case.

4.9 Multi antibiotic resistance index of various pathogens

4.9.1 Common resistance phenotype of *E. coli* for patients

E. coli shows most of the common phenotypes, having MAR index lowest like 0.05 and highest 0.32 , (Table 15).

Resistance Phenotype	No.	MAR index
AZM, SMX	2	0. 05
AZM, GM	2	0.06
CEF, CTX, CE,CIP,NIT,VAC	2	0.15
AMC,AZM,CFM,CTX,CRO,CE,LFX,	6	0.19
AZM,CE,GM,LZD	4	0.11
AZM,CFM,LZD	2	0.11
AZM	4	0.03
AZM,CFM,CRO,CT,NIT	4	0.14
AZM,CT,TE	2	0.08
AZM,NAL,TE	2	0.08
AMC,AZM,CEF,CRO,CT,LFX,NAL,TE	2	0.21
AZM,CEF,CFM,CAZ,CRO,CIP,CT,LFX,NAL,NET,SMX,TE	2	0.32
AMX,AZM,CEF,CFM,CRO,CIP,LFX,NAL,SMX,TE	6	0.26
AMC,AZM,CT,NAL,SMX	2	0.14
AZM,C,NAL,SMX,TE	5	0.14
AZM,CEF,CFM,CRO,SMX	2	0.14
AZM,CEF,CFM,CAZ,CRO,CIP,GM,LFX,SMX	3	0.24
AMX,CRO,CIP,CT,LFX,NAL,TE	2	0.19
CEF,CFM,CTX,CRO,CE	4	0.13
AZM,CEF,CFM,CRO,CE,GM,IPM,LFX,SMX	5	0.24
AMC,AZM,CEF,CFM,CTX,CRO,CE,IPM,LFX,SMX	5	0.27
AMK,AMC,AZM,CEF,CFM,CTX,CRO,CE,GM,TE	3	0.26
LFX,NIT	2	0.05
AZM,CREF,CFM,CAZ,CE,GM,NIT	2	0.18
AMX,AZM,CE,NIT	3	0.11
AMK,AZM,CEF,CFM,CAZ,CRO,LFX	2	0.18
AMK,AZM,GM	2	0.08
AMC,AZM,CEF,CFM,CAZ,CRO,CIP,LFX,TE	2	0.23
AZM,CFM,CAZ,CRO,CE,SMX,TE	2	0.19
AZM,CEF,CFM,CAZ,CRO,LFX	2	0.18
AZM,CEF,CFM,CAZ,CRO,GM,SMX	2	0.19

AZM,CTX,CRO,CE,GM,TE	2	0.16
AZM, VAC	2	0.05
AZM,CRO,SMX	2	0.08
AMX,AZM,CRO,CXM,CE,CIP,LFX,TE	2	0.21
CEF,CFM,CAZ,CRO,CE,GM,LFX,SMX	2	0.24
AZM,CFM,CT,SMX	2	0.12
AZM,CEF,CFM,CAZ,CRO,CE,CIP,GM,LFX,SMX,TE	3	0.29
AMK,AZM,CFM,CE,GM,SMX	2	0.16
AZM,CEF,CFM,CAZ,CRO,CE,TE	2	0.18
AZM,CE,CIP,GM,LFX,SMX,TE	2	0.18
AMC,AZM,GM	3	0.08
AZM,CE,NIT	2	0.08

Table 16: Common resistance phenotype and MAR index of E. coli

4.9.2 Common resistance phenotype of *S. aureus.*

Patients with *S.aureus* shows a common phenotype where MAR index 0.03 is the lowest and 0.22 is highest , (Table 17).

Resistance Phenotype	No.	MAR index
GM	2	0.03
AZM,TE	2	0.05
AMK, AZM,TE	3	0.08
AMK,AZM,TE,VAC	3	0.11
AZM,CEF,CRO,CT,GM,SMX,TE	4	0.19
AMX,LZD	3	0.05
AMK,AZM,CEF,CFM,CRO,CE,GM,SMX	3	0.22
CEF,CFM,CE,CT,ERY,TE	4	0.16
AZM,CRO,CE	2	0.8

Table 17: Common resistance phenotype and MAR index of S. aureus patients

4.9.3 Common resistance phenotype of *Streptococcus spp* .

Streptococcus spp shows MAR index of 0.03in 3 phenotypes, 0.19 in 3 phenotypes & 0.24 in 2 of the common phenotypes, (Table 18).

Common Resistance Phenotype	No.	MAR index
AZM,CFM,CRO,CIP,CT,GM,IPM,LFX,SMX	2	0.24
AZM,CEF,CAZ,CE,LZD,SMX,VAC	3	0.19
TE	3	0.03

Table 18: Common resistance phenotype and MAR index of Streptococcus spp. patients

4.9.4 Common resistance phenotype of *S. Typhi*

The common resistance phenotype of *S.typhi* consists of 0.03 in most patients MAR index and then it's 0.06, (Table 19).

Common Resistance Phenotype	No.	MAR index
AZM	5	0.03
AZM, NIT	9	0.06
GM	3	0.03
NAL	2	0.03
AZM, GM	2	0.06

Table 19: Common resistance phenotype and MAR index of S. Typhi patients

4.9.4 Common resistance phenotype of *Acinetobacter spp.*

MAR index of *Acinetobacter spp.* is 0.3 in two of the common phenotypes, (Table 20).

Common Resistance Phenotype	No.	MAR index
AMK,AZM,CFM,CTX,CE,NIT	2	0. 17
AZM	2	0.03
NAL	4	0.03
CFM,CT,DO,ERY	3	0.11
AZM,CEF,CRO,CE,CT,GM	2	0.16

Table 20: Common resistance phenotype and MAR index of Acinetobacter spp.

4.9.6 Common resistance phenotype of *Pseudomonas spp.* patients

Pseudomonas spp. shows the following pattern of resistance where MAR index of two patterns are 0.18 and it starts from 0.08 to 0.22 in some phenotypes of this pattern, (Table 21).

Common Resistant Phenotype	No.	MAR index
AMC,AZM,CRO,NAL,NIT,SMX,TE	4	0. 18
AZM,CEF,CAZ,CRO,CE,NIT,SMX	2	0.18
AZM, CEF, CRO, CT, NAL	3	0.13
AZM, CEF, CFM, CAZ, CE, CIP, LFX, SMX	2	0.22
AZM, NIT, SMX	2	0.08
AZM,CEF,CFM,CAZ,CRO,SMX	2	0.16
CEF, CFM, CAZ, CRO, CIP, CT, LFX,	2	0.19

Table 21: Common resistant phenotype and MAR index of Pseudomonas spp. patients

4.9.7 Common resistant phenotype of *Klebsiella ssp.* for patients

Klebsiella spp. exhibit resistance to the given pattern having MAR index 0.03 , (Table 22).

Common Resistant Phenotype	No.	MAR index
GM	3	0.03

Table 22: Common resistant phenotype and MAR index of Klebsiella.spp.

Chapter 5: Limitations

In thus study, we analyzed data from a single hospital. This study is also limited by only analyzing 1,890 samples of which merely 489 demonstrated positive microbial growth. In addition, some data in the hospital's database was lost in previous years because of COVID-19. Without this, data had limited availability and access.

Chapter 6: Conclusion

In the face of sharply increasing levels of multi-drug resistance among nosocomial bacterial pathogens, which are providing a growing worldwide reservoir for untreatable bacteria, these infections represent an intimidating challenge. However, the prevalence of these microorganisms creates a significant burden in terms of diagnosing and treating infections. Clinically multi-drug resistance (MDR) results in increased rates of patient morbidity and mortality leading to greater healthcare costs as well. Enhanced investment in R&D, collaboration across healthcare sectors at home and across international boundaries must be coupled with the development of global strategies to prevent misuse of antibacterial agents and monitor trends in resistance such that they can respond more effectively to threats posed by resistant pathogens. The global rise of multidrug-resistant organisms seriously threatens the continuity and safety of present-day health care services, as well as individual patient results. Thus, it is necessary to consider ways of proactively addressing these resistant infections and protecting the health of our populations.

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