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To Determine The Bacterial Phenotype Of Surgical Site Infections That Occurred In Indore.

Amit kumar Bharti[1], Dr.Harshada Shah[2],Dr. Kailash Jatav[3],Komal Singh[4], Navdeep[5]

Amit kumar Bharti, Ph.D Scholar, Dept. of Microbiology, Malwanchal University, Indore, Madhya Pradesh
Dr.Harshada Shah, Professor, Dept. of Microbiology, Malwanchal University, Indore, Madhya Pradesh
Dr. Kailash Jatav, Assistant Professor, Dept. of Microbiology, LNCT Medical College, Indore, MP
Komal Singh, Ph.D Scholar, Dept. of Microbiology, Malwanchal University, Indore, Madhya Pradesh

Abstract

Background: Although, the pathogens of surgical site infections (SSIs) are same, a shift from naïve bacteria to drug-resistant organisms has been observed, resulting in a worse prognosis for patients and increased costs for the Health Systems.

Aim: The aim of the present study is to determine the bacterial flora associated with SSI with their antimicrobial susceptibility (AST) pattern & resistance mechanisms in a tertiary care hospital.

Methods: The research is experimental in nature, but it also contains a descriptive element. Both of these are being looked into. Before it was carried out in Indore, it was given the go light by the Institutional Ethics Committee of the Index Medical College Hospital and Research Centre (IMCH&RC). Patients from the fields of surgery, obstetrics, and orthopedics who suffered postoperative wound infections participated in this study. 248 surgical patients participated in the research study.

Results: 50% of the GNB isolates were positive for ESBL. ESBL positivity was significantly more prevalent (>50%) in E. coli, Klebsiella spp., and Enterobacter spp. than in Acinetobacter spp. (1%). Similarly, 89% of E. coli, Klebsiella spp., and Enterobacter spp.were MDR, with Acinetobacter spp. There was a variation in susceptibility status according to specific bacterial genus and antimicrobial agent, but in general, fifty five percent of the GNB isolates that were tested showed resistance to the antimicrobials that were included in the first-line AST panel.

Conclusion: Klebsiella was discovered to be the most common type of bacterium responsible for SSI following abdominal procedures. Periodic observation of SSI will aid in the development of stringent standards, lowering the occurrence of SSI.

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I. Introduction:

Antibiotic resistance is responsible for 700,000 deaths worldwide each year [1,2]. SSI are defined as infections that occur 30 days after surgery with no implant, or within 1 year if an implant is placed and infection appears to be related to surgeries [3,4]. These are the most frequent type of healthcare-associated infection (HAI) observed on admission in low- and middle-income countries (LMICs). The incidence of SSIs ranges between 2.5-41.9%, with a significantly higher percentage in developing countries as compared to developed countries [3-6]. The patients of SSIs have 2-11 times greater risk of death in comparison to the patients having no SSI [7]. The number of incidences reported for SSI may be different across several countries due to the various systems applied for the epidemiological control of hospital related infection [8]. SSI can sometimes be superficial infections involving the skin only. Other surgical site infections are more serious and can involve tissues under the skin, organs, or implanted material [9]. Approximately one in 10 people who have surgery in LMICs acquire an SSI, SSI is reported as the second most common HAI in Europe and the United States of America [10]. Staphylococcus aureus, coagulase negative staphylococci (CONS), Enterococcus spp., Escherichia coli are still the most frequently isolated pathogens from SSIs. Staphylococcus aureus is the most frequently isolated organism in SSI, accounting for 15-20% of SSIs occurring in hospital. Other common isolates include gram-negative bacilli, coagulase-negative Staphylococci, Enterococcus spp., and Escherichia coli [4-6] Although, the pathogens of surgical site infections (SSIs) are same, a shift from naïve bacteria to drug-resistant organisms has been observed, resulting in a worse prognosis for patients and increased costs for the Health Systems [3-9]. Staphylococcus aureus and Escherichia coli are the most frequently isolated bacteria from SSI culture, with a resistance rate

ranging from 42.7 to 44.7% for S. aureus, and from 13.3 to 15.3% for E. coli [5]. Methicillin resistant S. aureus (MRSA) is an increasingly important pathogen that causes more than 50% of S. aureus hospital acquired infections in the US and Europe and presents challenges to treatment due to multiple antibiotic resistance [10,11].

Hence, the aim of the present study is to determine the bacterial flora associated with SSI with their antimicrobial susceptibility (AST) pattern & resistance mechanisms in a tertiary care hospital.

II. Materials & Methods:

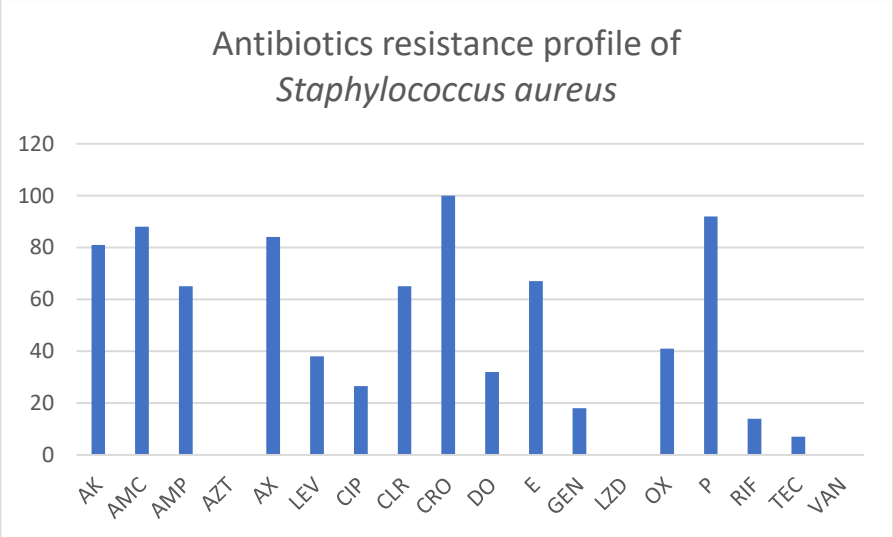
The research is experimental in nature, but it also contains a descriptive element. Both of these are being looked into. Before it was carried out in Indore, it was given the go light by the Institutional Ethics Committee of the Index Medical College Hospital and Research Centre (IMCH&RC). Patients from the fields of surgery, obstetrics, and orthopedics who suffered postoperative wound infections participated in this study. 248 surgical patients participated in the research study. Inclusion criteria: Patients who were over the age of 18 and who were planned to have emergency or elective laparotomies (clean-contaminated or contaminated surgeries) and who were willing to participate in the study after providing written informed permission were considered eligible. Those who do not cooperate, are morbidly obese, or are allergic to povidone iodine are not allowed to take part in the study. Pus exudate was collected for aerobic culture using two sterile cotton swabs. Pus was aspirated using a sterile syringe for anaerobic culture, and then it was inoculated onto Blood agar and McConkey agar, Nutrient agar, and Robertson cooked meat medium. Conventional microbiological methods were used to identify pure isolates from subculture plates. These methods studied colony morphology, Gram staining reactions, and biochemical properties. Catalase and oxidase tests, Hugh Lifson's oxidative fermentative agar, sugar fermentation media, indole, methyl red, Voges Proskauer, Simon's citrate, triple sugar iron agar, and Christensen's [10-19]. These procedures were carried out using sterile and aseptic precautions throughout the entire process. The samples were put through a battery of diagnostic procedures, including a direct microscopic examination of a Gram-stained smear, a preliminary identification based on colony morphology, a biochemical test for the characterization of species, and antibiotic sensitivity testing. Antibiotic resistance testing. The Clinical and Laboratory Standards Institute recommended using a modified Kirby-Bauer disc diffusion method to assess isolate antibiotic susceptibility. This determined which antibiotics would be most effective against the isolates (CLSI). All these procedures were carried out in order.

Statistical Analysis:

SPSS (Statistical Program for the Social Sciences) was used for statistical analysis. Antibiotic resistance was calculated by dividing the number of resistant isolates by the total number examined. We calculated the multiple antibiotic resistance (MAR) index for each isolate by dividing the number of antibiotics to which it was resistant by the total number of antibiotics tested. We employed Student's t-test and one-way analysis of variance (ANOVA) to compare continuous variables between two and more than two groups, respectively. The P0.05 value determined statistical significance.

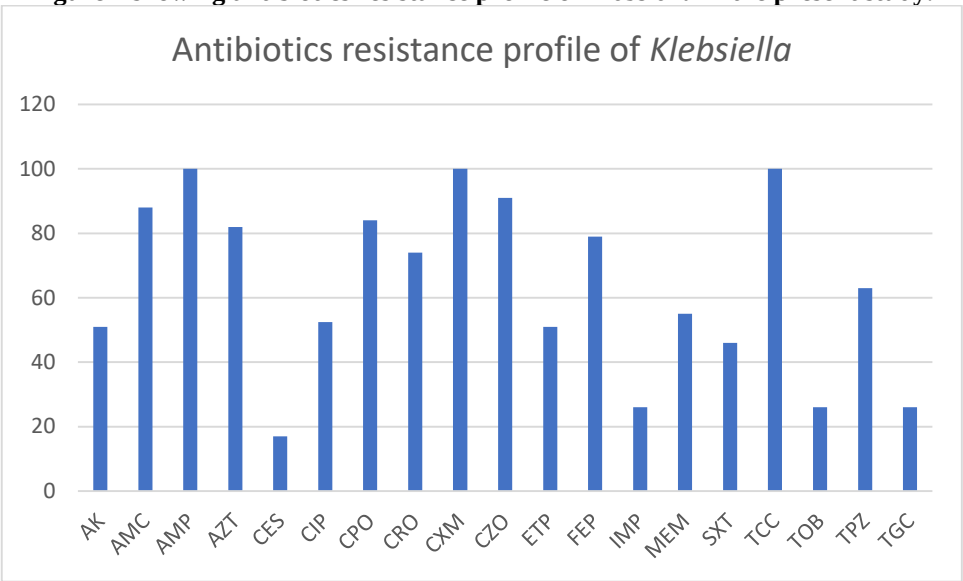
III. Results:

Figure 1 showing antibiotics resistance profile of *S. aureus* in the present study.



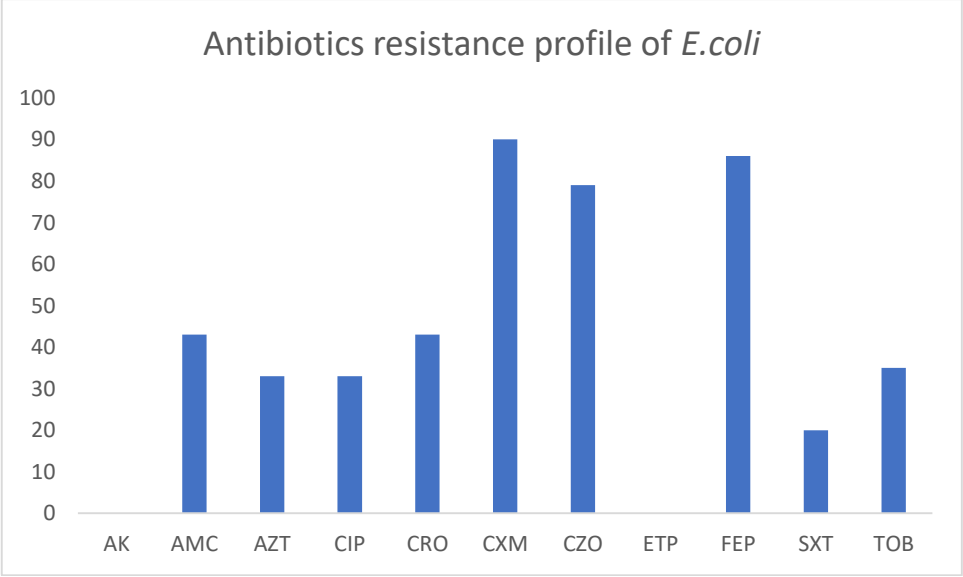
Note: AK, amikacin; AMC, amoxicillin/clavulanate; AMP, ampicillin; AZT, aztreonam; AX, amoxicillin; LEV, Levofloxacin; CIP, ciprofloxacin; CLR, clarithromycin; CRO, ceftriaxone; DO, doxycycline; E, erythromycin; GEN, gentamycin; LZD, linezolid; OX, oxacillin; P, penicillin; RIF, rifampin; TEC, teicoplanin; VAN, vancomycin.

Figure 2 showing antibiotics resistance profile of *Klebsiella* in the present study.



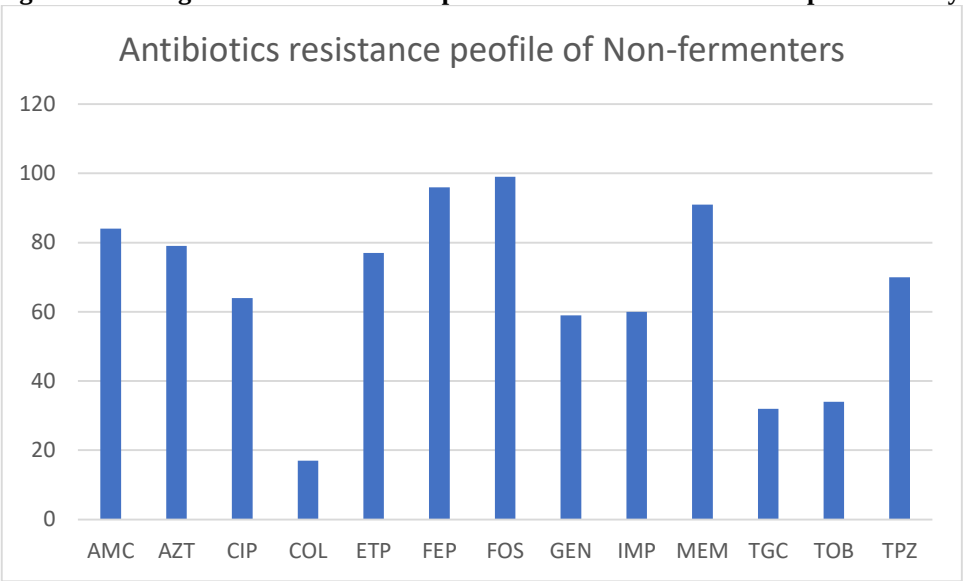
Note: AK, amikacin; AMC, amoxicillin/clavulanate; AMP, ampicillin; AZT, aztreonam; CES, cefoperasone sulbactam; CIP, ciprofloxacin; CPO, cefpirome; CRO, ceftriaxone; CXM, cefuroxime; CZO, cefazoline; ETP, ertapenem; FEP, cefepim; IMP, imipenem; MEM, meropenem; SXT, trimethoprim/sulfamethoxazole; TCC, ticarcillin/clavulanate; TGC, tigecycline; TOB, tobramycin; TPZ, piperacillin-tazobactam.

Figure 3 showing antibiotics resistance profile of *E. coli* in the present study.



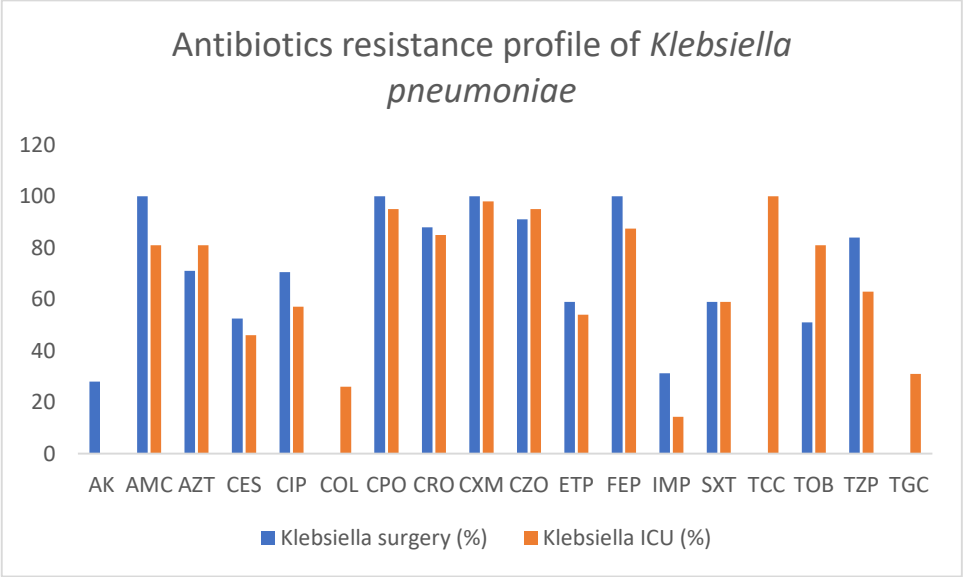
Note: AK, amikacin; AMC, amoxicillin/clavulanate; AZT, aztreonam; CIP, ciprofloxacin; CRO, ceftriaxone; CXM, cefuroxime; CZO, cefazoline; ETP, ertapenem; FEP, cefepim; SXT, trimethoprim/sulfamethoxazole; TOB, tobramycin.

Figure 4 showing antibiotics resistance profile of non-fermenters in the present study.



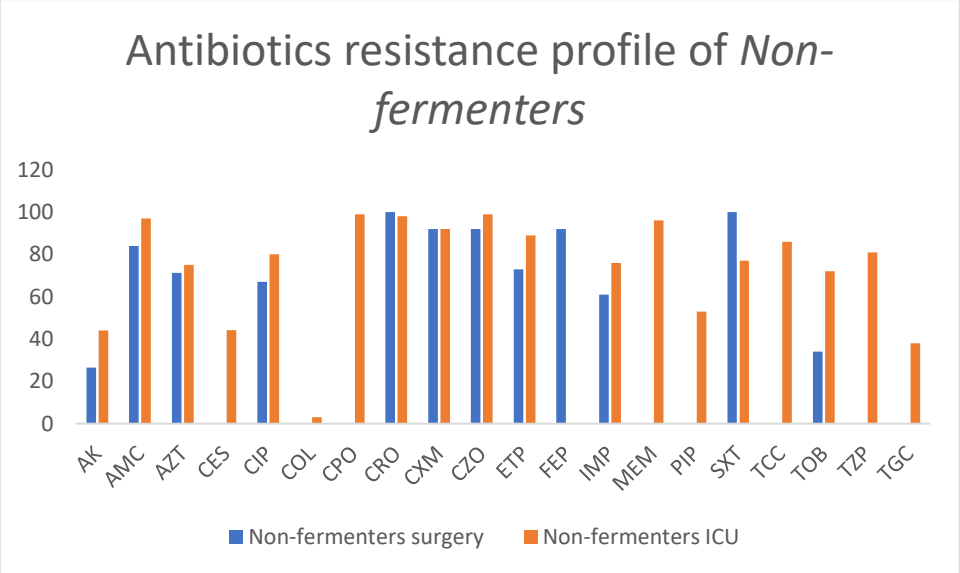
Note: AMC, amoxicillin/clavulanate; AZT, aztreonam; CIP, ciprofloxacin; CLR, clarithromycin; COL, colistin; ETP, ertapenem; FEP, cefepim; FOS, fosfomycin; GEN, gentamycin; IMP, imipenem; MEM, meropenem; TGC, tigecycline; TOB, tobramycin; TPZ, piperacillin-tazobactam.

Figure 5 showing antibiotics resistance profile of *Klebsiella pneumoniae*.



Note: AK, amikacin; AMC, amoxicillin/clavulanate; AMP, ampicillin; AZT, aztreonam; CES, cefoperasone sulbactam; CIP, ciprofloxacin; CPO, cefpirome; CRO, ceftriaxone; CXM, cefuroxime; CZO, cefazoline; ETP, ertapenem; FEP, cefepim; IMP, imipenem; MEM, meropenem; SXT, trimethoprim/sulfamethoxazole; TCC, ticarcillin/clavulanate; TGC, tigecycline; TOB, tobramycin; TPZ, piperacillin-tazobactam.

Figure 6 showing antibiotics resistance profile of *non-fermenters.*



The fifth and sixth objective of the present study were to study the phenotypic & genotypic resistance mechanisms among selected resistant isolates from SSIs.

Table 1 showing the phenotypic status of ESBL positivity and Multi-drug resistant (MDR) of bacteria isolated from blood samples.

Bacterial pathogens	ESBL-negative		ESBL-Positive		Non-MDR		MDR	
	N	%	N	%	N	%	N	%
Klebsiella (n=93)	41	45	52	55	8	12	46	88
E. Coli (n=69)	28	39	42	61	5	9	56	92
Enterobacter spp. (n=62)	24	37	39	63	1	2	38	98
Klebsiella spp. (n=48)	48	99	1	1	0	0	1	100

The isolated Klebsiella strains were found to be 100% resistant to ampicillin, cefuroxime and ticarcillin/ clavulanate, and resistant to cefazolin (91%), amoxicillin/ clavulanate (88%), cefpirome (84%), aztreonam (82%), cefepime (79%), piperacillin with tazobactam (63 %), ciprofloxacin (52.5%), amikacin (51 %) and sulfametoxazole with trimethoprim(46 %). A low level of resistance was observed to cefoperasone-sulbactam (17 %), imipenem (26 %), tigecycline (26 %) and tobramycin (26 %).

An increased level of resistance to cefuroxime (90 %), cefepime (86%), and cefazoline (79%) was detected after completing antimicrobial susceptibility tests on the isolated E. coli strains. There was shown to be a moderate resistance to amoxicillin/clavulanate (43%), ceftriaxone (43%), aztreonam (33%), and ciprofloxacin (33%). The E. coli bacteria tested showed only a moderate level of resistance to trimethoprim/sulfamethoxazole (20%), while showing no resistance to amikacin or ertapenem.

The bacteria that did not digest glucose exhibited a significant level of resistance to antibiotics. For instance, the resistance to fosfomycin was quite strong (99%), as was the resistance to cefepime (96 %), meropenem (91 %), amoxicillin/clavulanate (84%), aztreonam (79%), and ertapenem (77%). There was a high level of resistance to piperacillin-tazobactam (70%), ciprofloxacin (63 %), imipenem (60%), and gentamycin (59%), but there was a low level of resistance to tobramycin (34%), tigecycline (32 %), and colistin (17%). In general, the non-fermenting bacteria collected from patients in intensive care units had a higher level of resistance than those collected from surgical wards.

There was a significant difference in the antibiotic resistance of Klebsiella strains between the surgical wards and the ICU in terms of imipenem [31.20 percent in ICU vs. 14.30 percent in surgical wards, risk ratio (RR) = 2.182], piperacillin with tazobactam (62.80 vs. 83.30%, RR = 0.754), tobramycin (80.00 vs. 50.00 percent, In terms of resistance to cefuroxime, cefpirome, and amoxycillin/clavulanate, there were no discernible changes between the groups. We are able to see that more than fifty percent of the ICU strains were resistant to practically every drug.

Ceftriaxone (100%), penicillin (92%), amoxicillin/clavulanate (88 %), amikacin (81%), and amoxicillin (84%) were all ineffective against the highly resistant S. aureus strains. There was a forty percent resistance to cefoxitin. The following antibiotics were shown to have low resistance rates: levofloxacin (38 %), doxycycline (32 %), gentamycin (18 %), tigecycline (7%), and teicoplanin (6.90%).

50% of the bacterial isolates were positive for ESBL. ESBL positivity was significantly more prevalent (>50%) in Klebsiella spp., and non-fermenters (3 %). Similarly, 89% of E. coli, Klebsiella spp., and non-fermenters were MDR.

IV. Discussion:

This study examines SSI-associated bacterial flora and their antimicrobial susceptibility (AST) patterns and resistance mechanisms in a tertiary care hospital. To maximize surgical prophylactic antibiotic therapy, SSI-recovered bacteria's antibiotic resistance must be known. This prevents the selection of multi-antibiotic-resistant bacteria. Drug-resistant bacteria emerge naturally, although non-rational antibiotic therapy can accelerate the process. Hospital ICUs are known to propagate multi-resistant pathogens. This underscores the need of comparing ICU resistance to surgical ward resistance.

SSI occurs in 22.41% of clean-contaminated operations. Surgical site infection in clean and clean-contaminated instances was studied by [20-23]. Our analysis found 27% SSI prevalence. The drape and no drape groups had similar hospital stays. The iodine-impregnated incise drape did not significantly reduce hospital stays in comparable studies [24-28].

Emergency surgeries have 2.5 times the SSI risk of planned surgery. According to [29-31], the length of the procedure increases the risk of surgical site infection. Our study found that SSI rates are considerably greater in surgical operations that last longer than three hours. Another study [32] employing identical methodology found comparable results.

This study found medication resistance in ICU and surgery ward microorganisms. Antibiotic use in ICUs raises bacterial resistance. Low cephalosporin resistance. ICU and surgery ward cefepime therapy differed most. These findings confirm a prior study that demonstrated modest differences in cephalosporin-resistant bacteria between intensive care units (ICUs) and surgical wards for the 1st and 2nd generations, but much higher variances for the 4th generation (84.2 vs. 69.82% for cefepime) [23, 24]. Intensive care unit Klebsiella may have spread to surgical wards. This may be because many surgical patients go straight from the operating room to the intensive care unit, where they stay for a few days before returning to the surgical ward. The high ICU prescription rate may have caused carbapenem resistance (32.2 vs. 14.8% for imipenem) [23]. Beta-lactam antibiotics cause resistance after a few years [25,26].

We observed that non-fermenting bacteria were more resistant to antibiotics, especially fourth-generation cephalosporins, penicillins with inhibitors, and carbapenems. Meropenem was 90.00% resistant, imipenem 59.10%. Meropenem prescriptions explain resistance discrepancy. Cephalosporin resistance was higher than aminoglicoside resistance (57.10 percent to gentamycin). Abdominal surgery still requires prophylactic cephalosporins [27]. Antibiotic minocycline is safe. It prevents Acinetobacter baumannii better now [28].

Klebsiella displayed less resistance variation than non-fermenting bacteria. Many ICU patients are immunocompromised [29]. Hence, they may be more sensitive to environmental pathogens such Acinetobacter [30] or Fusarium [31]. Immune patients cannot contract common environmental pathogens. Acinetobacter is found in the intensive care unit, while Pseudomonas is found in several hospital rooms (ICU). Antibiotics make ICU bacteria more resistant.

MRSA slows surgical wound healing [32]. 40% of S. aureus isolates were MRSA. Hospitals spread MRSA easily. Due to antibiotic overuse, MRSA may evolve from MSSA [33,34]. Our study found 20.29% MRSA prevalence (40% x 50.72%). MRSA (40%), S. aureus (50.72%). This matches SSI MRSA prevalence of 28.50% [35].

Few susceptible S. aureus strains exhibit mecA-positive cells [36]. MSSA colonisation is common at admission [36]. Acne-prone people have S. aureus [37]. 48-hour cefoxitin or oxacillin exposure isolates these MRSA subpopulations [38]. SSI MRSA prevalence may approach 20.29 percent [39].

Only a few Staphylococcus bacteria secreted -lactamases in the study samples, suggesting that only a few could. Ciprofloxacin promotes quinolone resistance [40]. The current analysis demonstrated 26.50% ciprofloxacin resistance despite numerous surgeons using quinolones prophylactically. Rifampin's 13.80% resistance rate is because it treats tuberculosis. 15.50% of S. aureus strains were rifampin-resistant [41].

Epidemiology and strain homology measure antibiotic resistance using disc diffusion inhibition zone widths [42]. Antibiotic resistance analysis frequently emphasises resistance rather than inhibition zone diameter. Most methods can't use this variable's continuity. Cluster analysis generates homology trees from continuous data to show isolate relatedness [43].

Staphylococcus resistance cluster dendrograms clearly identify MRSA strains from sensitive bacteria. Hierarchical MRSA resistance suggests more genes. 40% of procedures have postoperative infections. Gram-negative bacteria and S. aureus cause most SSIs. This high prevalence may be due to the high load of S. aureus in air flora, patient colonization at admission, and other unidentified perioperative factors. ICU bacteria are carbapenem-resistant. ICUs should not use these agents to prevent resistance. Antibiotic resistance-optimized anti-infective therapy would replace inefficient drugs with more effective ones, improving medical care. This study's surgical antibiotic guidelines can aid infection control. This reduces SSI risk and ensures successful anti-infectious use.

V. Conclusion:

It is important that efforts be made to shorten the duration of surgical procedures while keeping the same level of care. E. coli and Klebsiella was discovered to be the most common type of bacterium responsible for SSI following abdominal procedures. Periodic observation of SSI will aid in the development of stringent standards, lowering the occurrence of SSI. The information gleaned from this study will assist the infection control team in developing effective guidelines for antibiotic therapies.

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