

Bacterial Patterns and Antibiotic Resistance Profiles at Advent Hospital Bandar Lampung, January–June 2025

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ABSTRACT

Antimicrobial resistance (AMR) poses a significant global health threat, necessitating local surveillance to guide empirical therapy and antimicrobial stewardship. This study aimed to characterize the distribution of bacterial pathogens and their antibiotic resistance profiles at Advent Hospital Bandar Lampung. A descriptive cross-sectional study was conducted using 171 clinical specimens collected from January to June 2025. Bacterial identification and antimicrobial susceptibility testing were performed in accordance with CLSI guidelines. The 171 specimens, 91 yielded bacterial growth, dominated by Gram-negative organisms—specifically *Klebsiella pneumoniae* and *Escherichia coli*. Gram-negative isolates exhibited high resistance to ampicillin and third-generation cephalosporins, whereas meropenem, amikacin, cefoperazone-sulbactam, and piperacillin-tazobactam remained effective. Gram-positive pathogens, primarily *Staphylococcus aureus*, showed substantial β -lactam resistance but maintained susceptibility to vancomycin, linezolid, and gentamicin. Critical phenotypes identified included carbapenem-resistant *Acinetobacter baumannii* (CRAB), third-generation cephalosporin-resistant Enterobacterales (3GCRE), and methicillin-resistant *S. aureus* (MRSA). The high prevalence of AMR in this setting underscores the urgent need to update empirical treatment guidelines, strengthen antimicrobial stewardship programs, and enhance infection prevention and control measures.

Key Messages:

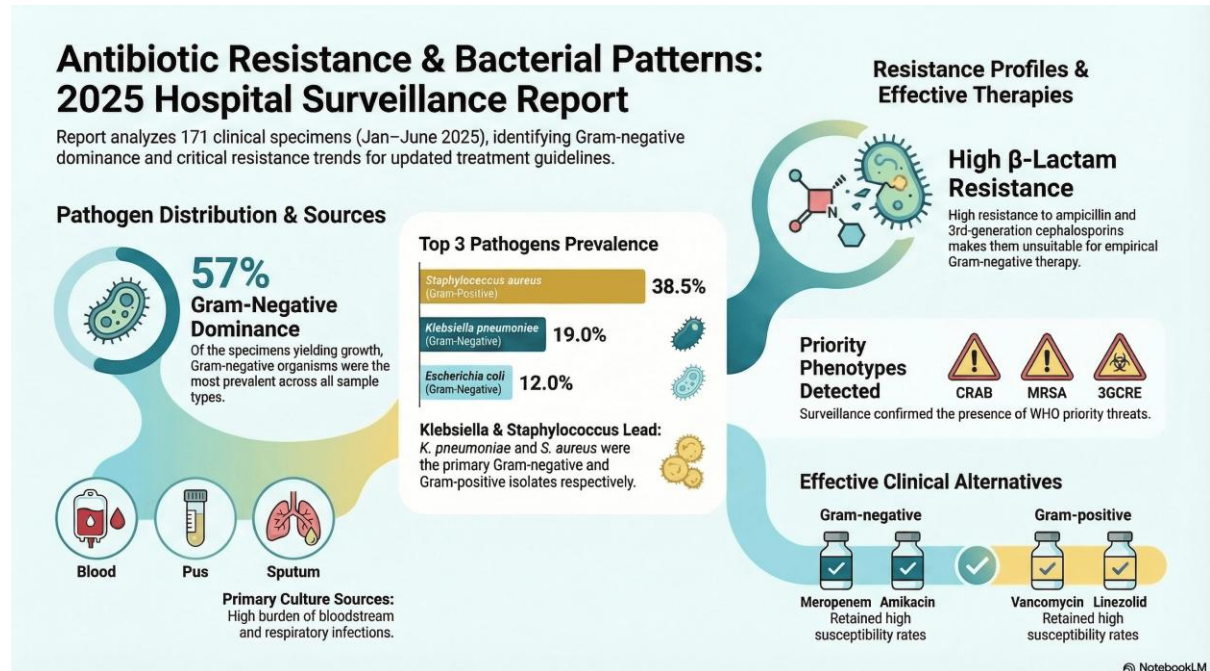
- The results are expected to serve as a foundation for updating empirical therapy guidelines, strengthening the Antimicrobial Stewardship Program (ASP), and evaluating the Infection Prevention and Control (IPC).
- The results are also expected to reduce infection rates, length of stay, mortality, and healthcare costs in the hospital.

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GRAPHICAL ABSTRACT



INTRODUCTION

Infections caused by pathogenic bacteria are among the most common health problems encountered in hospitals, occurring both as community-acquired infections and healthcare-associated infections (HAIs). These infections have a significant impact on increasing morbidity, mortality, length of hospital stay, and healthcare costs [1]. One of the greatest challenges in infection management is the rising bacterial resistance to antibiotics. Antimicrobial resistance (AMR) occurs when bacteria undergo changes that render antibiotics ineffective in killing or inhibiting their growth. This phenomenon poses a serious global threat, including in Indonesia, as it can lead to limited treatment options, reliance on last-line antibiotics, and an increased risk of treatment failure [2].

Moreover, the burden of antimicrobial resistance (AMR) continues to grow and has been linked to 4.95 million deaths globally in 2019, with 1.27 million of those deaths directly attributed to AMR. This number surpasses several other priority infectious diseases and highlights the urgent need for AMR control in healthcare settings, particularly in hospitals, where antibiotic selective pressure is high [3]. As healthcare facilities, hospitals play a critical role in controlling antibiotic resistance. The patterns of infectious pathogens and their resistance profiles can vary between hospitals and may change over time depending on patient characteristics, antibiotic usage, and infection control policies. Therefore, regular monitoring and analysis of pathogen patterns and antibiotic resistance are essential as a basis for developing antibiotic stewardship policies and infection prevention and control (IPC) strategies [4, 5].

Multinational studies in healthcare settings have shown high levels of resistance in key nosocomial pathogens: carbapenem-resistant *Acinetobacter baumannii* ($\approx 70\%$), carbapenem-resistant *Pseudomonas aeruginosa* ($\approx 36\%$), and carbapenem-resistant Enterobacterales (e.g., *Klebsiella pneumoniae* $\approx 22\%$ and *Escherichia coli* $\approx 18\%$). The burden of MRSA and penicillin-resistant *Streptococcus pneumoniae* also remains significant. These patterns, when reflected in the local hospital context, have the potential to increase treatment failure, length of hospital stay, and healthcare costs [6].

At the global level, the World Health Organization (WHO), through the Global Antimicrobial Resistance and Use Surveillance System (GLASS), emphasizes the importance of standardized surveillance of priority pathogens and antibiotic consumption to guide clinical policies and stewardship programs. The latest GLASS report highlights inter-country variations in antibiotic use and resistance, as well as the need to improve data quality at healthcare facilities. These findings are particularly relevant for hospitals, as local pathogen patterns and susceptibility play a crucial role in the success of empirical therapy [7].

In Indonesia, the commitment to AMR control is reinforced through Ministry of Health Regulation

No. 8 of 2015 on the Antimicrobial Resistance Control Program (PPRA), and Regulation No. 27 of 2017 on Infection Prevention and Control (IPC), which require hospitals to conduct pathogen surveillance and susceptibility testing, antibiotic use audits, and regular reporting. The National Action Plan (NAP) for AMR Control also emphasizes cross-sectoral collaboration and the strengthening of surveillance systems. Consistent implementation at the hospital level is key to ensuring the availability of regular antibiograms as a basis for rational empirical therapy selection [8, 9, 10].

Although several AMR studies have been conducted in Indonesia, available evidence remains outdated, unit-specific, or derived from hospitals with different epidemiological characteristics [6, 7]. To date, no updated and hospital-specific resistance data have been published for Advent Hospital Bandar Lampung, creating a gap in locally relevant evidence for empirical antibiotic selection [8].

This study provides the first six-month, institution-specific surveillance of bacterial patterns and antibiotic resistance at Advent Hospital Bandar Lampung. It includes both Gram-negative and Gram-positive pathogens and identifies WHO priority AMR organisms such as CRAB, MRSA, and 3GCRE [3, 7]. These findings provide new local evidence to support empirical therapy guidelines, antimicrobial stewardship, and infection prevention and control. Therefore, this study aims to describe bacterial patterns and analyze antibiotic resistance profiles from January to June 2025.

METHODS

Sample Collection and Processing

This study employed a descriptive cross-sectional, laboratory-based surveillance design, conducted over a six-month period from January to June 2025 at Advent Hospital Bandar Lampung, Indonesia. Samples were collected from both inpatient and outpatient populations. All specimens were obtained in sterile containers by designated medical personnel and promptly processed in the microbiology laboratory. Specimen preservation and storage varied depending on specimen type and clinical need. For example, blood, urine, and sputum samples were retained until analysis and physician consultation were completed, while high-value specimens such as postoperative samples or body fluids were kept for 7–10 days. Demographic and clinical information—including sex, age, ward, date of collection, specimen type, and other relevant details—were recorded at the time of sampling.

A total sampling approach was applied, including all 171 specimens submitted for culture from January to June 2025. Bacterial isolates used in this study were obtained from these samples and processed following CLSI guidelines [11].

Blood samples collected from adult patients (10–20 mL) and pediatric patients (5–10 mL) were inoculated into brain heart infusion (BHI) broth bottles and monitored for bacterial growth for up to seven days using an automated blood culture system, following standard CLSI procedures [11]. Positive blood cultures were subcultured on blood agar (Oxoid™), nutrient agar (Oxoid™), and MacConkey agar (Oxoid™) plates [11].

Urine samples (20–30 mL) collected in sterile 50 mL plastic containers were streaked on MacConkey agar (Oxoid™), blood agar (Oxoid™), cysteine lactose electrolyte-deficient (CLED) agar (Oxoid™), and nutrient agar (Oxoid™), according to CLSI recommendations [11]. Pus or swab samples were inoculated on nutrient agar (Oxoid™), blood agar (Oxoid™), and MacConkey agar (Oxoid™) [11]. Sputum specimens (2–5 mL) were collected in sterile containers and streaked on chocolate agar (Oxoid™), blood agar (Oxoid™), nutrient agar (Oxoid™), and MacConkey agar (Oxoid™), also following CLSI guidelines [11].

All specimens except blood were processed on the same day as collection. The inoculated agar plates were incubated at 35°C for 24–72 hours and subsequently subcultured on nutrient agar to ensure purity. Bacterial identification was performed based on colony morphology, biochemical reactions, and Gram staining characteristics according to CLSI standards [11].

Antibiotic Resistance Testing

Antibiotic resistance testing was carried out on all confirmed bacterial isolates using the Kirby–Bauer disk diffusion method on Mueller–Hinton agar, following the guidelines of the Clinical and Laboratory Standards Institute. Freshly isolated colonies were suspended in sterile saline to achieve turbidity

equivalent to 0.5 McFarland standard. A sterile cotton swab was then used to evenly inoculate the bacterial suspension onto the surface of the agar plate. Antibiotic discs were gently placed on the agar using sterile forceps, ensuring uniform contact between the discs and the medium. The plates were incubated at $35 \pm 2^\circ\text{C}$ for 18–24 hours under aerobic conditions. After incubation, the zones of inhibition surrounding each antibiotic disc were measured in millimeters using a calibrated ruler. The results were interpreted as Sensitive (S), Intermediate (I), or Resistant (R) according to CLSI criteria [11]. Because antibiotic resistance testing panels were determined based on CLSI recommendations, clinical relevance, and laboratory resource availability, not all isolates were tested against all antibiotics. Therefore, *n* values in tables represent the actual number of isolates tested per antibiotic [11, 12, 13].

Data Processing and Analysis

This study employed a descriptive cross-sectional (laboratory-based surveillance design). Bacterial isolates were categorized as susceptible, intermediate, or resistant in accordance with the interpretive standards outlined by the Clinical and Laboratory Standards Institute. Data were analyzed using descriptive statistical methods, including the frequency (%), species distribution, percentage of resistance per antibiotic and per species, and identification of WHO priority AMR pathogens including CRAB, 3GCRE, and MRSA.

CODE OF HEALTH ETHICS

This study was approved by the Health Research Ethics Committee of Malahayati University with approval number 5066/EC/KEP-UNMAL/X/2025.

RESULTS

Characteristics Based on Sample Type

A total of 171 clinical specimens were examined, and approximately half yielded bacterial growth. Gram-negative organisms were slightly more common than Gram-positive isolates. The sample distribution was dominated by blood cultures, followed by pus and sputum, indicating that most suspected infections in this period were bloodstream and soft-tissue related. Figure 2 highlights this pattern clearly, showing that blood was the primary specimen type submitted for culture, with pus and sputum contributing substantially to the overall diagnostic workload.

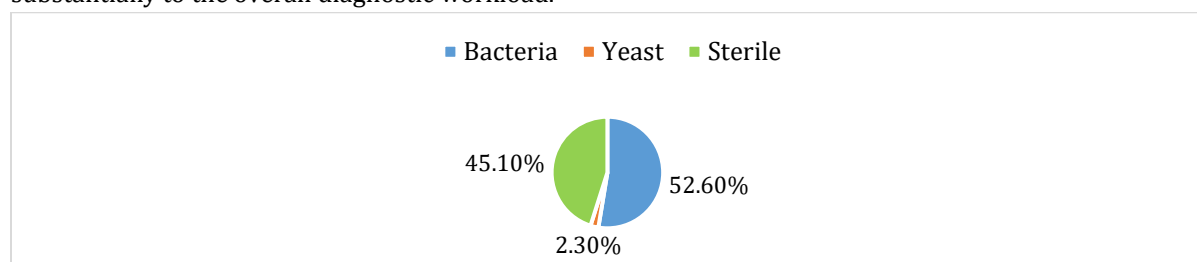


Figure 1. Culture Sample

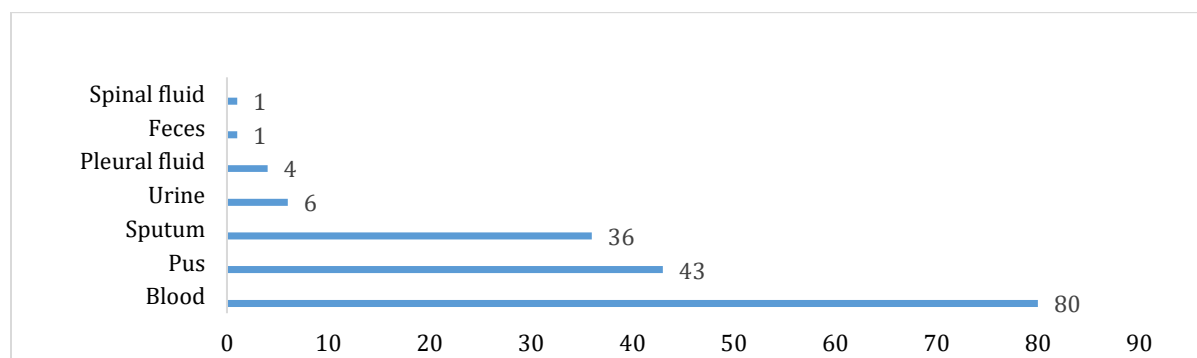


Figure 2. Types of Culture Samples

Characteristics Based on Bacterial Types

The bacterial distribution in this study was dominated by Gram-negative organisms, with *Klebsiella pneumoniae* emerging as the most common pathogen. This species appeared consistently across multiple specimen types, particularly respiratory samples, indicating its important role in lower-respiratory and hospital-associated infections. Other Gram-negative organisms—such as *Escherichia coli* and various *Acinetobacter* species—also contributed substantially, especially in pus, sputum, and urine specimens, reflecting their involvement in soft-tissue and urinary infections. Overall, the data suggest that respiratory and soft-tissue infections were the major contributors to the bacterial burden in this hospital during the study period, with *K. pneumoniae* representing the highest-impact pathogen among Gram-negative bacteria.

Table 1. Gram-Negative Bacteria

| No | Gram-Negative Bacteria | Types of Sample | | | | | Sum | % |
|-------|-----------------------------------|-----------------|--------|-------|-------|---------------|-----|-----|
| | | Pus | Sputum | Blood | Urine | Pleural Fluid | | |
| 1 | <i>Klebsiella pneumoniae</i> | 0 | 9 | 1 | 0 | 0 | 10 | 19 |
| 2 | <i>Escherichia coli</i> | 4 | 0 | 0 | 2 | 0 | 6 | 12 |
| 3 | <i>Acinetobacter sp</i> | 1 | 1 | 1 | 0 | 0 | 4 | 7.7 |
| 4 | <i>Klabsiella ornithinolytica</i> | 3 | 1 | 0 | 0 | 0 | 4 | 7.7 |
| 5 | <i>Burkholderia cepacian</i> | 3 | 1 | 0 | 0 | 0 | 4 | 7.7 |
| 6 | <i>Acinetobacter baumannii</i> | 1 | 2 | 0 | 0 | 0 | 3 | 5.8 |
| 7 | <i>Proteus mirabilis</i> | 3 | 0 | 0 | 0 | 0 | 3 | 5.8 |
| 8 | <i>Raoultella ornithinolytica</i> | 3 | 0 | 0 | 0 | 0 | 3 | 5.8 |
| 9 | <i>Enterobacter aerogenes</i> | 2 | 1 | 0 | 0 | 0 | 3 | 5.8 |
| 10 | <i>Pseudomonas aeruginosa</i> | 0 | 2 | 0 | 0 | 0 | 2 | 3.8 |
| 11 | <i>Moraxella sp</i> | 0 | 0 | 1 | 0 | 0 | 1 | 1.9 |
| 12 | <i>Enterobacter cloacea</i> | 0 | 0 | 0 | 0 | 1 | 1 | 1.9 |
| 13 | <i>Serratia plymuthica</i> | 1 | 0 | 0 | 0 | 0 | 1 | 1.9 |
| 14 | <i>Enterobacter agglomerans</i> | 1 | 0 | 0 | 0 | 0 | 1 | 1.9 |
| 15 | <i>Klebsiella ozaenae</i> | 1 | 0 | 0 | 0 | 0 | 1 | 1.9 |
| 16 | <i>Serratia odorifera</i> | 1 | 0 | 0 | 0 | 0 | 1 | 1.9 |
| 17 | <i>Morganella morganii</i> | 0 | 1 | 0 | 0 | 0 | 1 | 1.9 |
| 18 | <i>Serratia marcescens</i> | 0 | 1 | 0 | 0 | 0 | 1 | 1.9 |
| 19 | <i>Chromobacterium violaceum</i> | 0 | 1 | 0 | 0 | 0 | 1 | 1.9 |
| 20 | <i>Citrobacter youngae</i> | 0 | 0 | 0 | 1 | 0 | 1 | 1.9 |
| Total | | 24 | 20 | 3 | 3 | 1 | 52 | 100 |

Table 2. Gram-Positive Bacteria

| No | Gram-Positive Bacteria | Types of Sample | | | | | Sum | % |
|-------|--|-----------------|--------|-------|-------|---------------|-----|------|
| | | Pus | Sputum | Blood | Urine | Pleural Fluid | | |
| 1 | <i>Staphylococcus aureus</i> | 9 | 1 | 3 | 1 | 1 | 15 | 38.5 |
| 2 | <i>Staphylococcus xylosus</i> | 0 | 0 | 4 | 0 | 0 | 4 | 10 |
| 3 | <i>Staphylococcus intermedius</i> | 0 | 1 | 1 | 1 | 0 | 3 | 7.7 |
| 4 | <i>Staphylococcus epidermidis</i> | 1 | 2 | 0 | 0 | 0 | 3 | 7.7 |
| 5 | <i>Staphylococcus sciuri</i> | 1 | 1 | 0 | 0 | 0 | 2 | 5.1 |
| 6 | <i>Staphylococcus lentus</i> | 0 | 2 | 0 | 0 | 0 | 2 | 5.1 |
| 7 | <i>Aerococcus viridans</i> | 1 | 0 | 0 | 1 | 0 | 2 | 5.1 |
| 8 | <i>Staphylococcus capitis</i> | 0 | 1 | 0 | 0 | 0 | 1 | 2.6 |
| 9 | <i>Staphylococcus capitis subsp. Capitis</i> | 0 | 0 | 1 | 0 | 0 | 1 | 2.6 |
| 10 | <i>Staphylococcus cohnii</i> | 0 | 1 | 0 | 0 | 0 | 1 | 2.6 |
| 11 | <i>Staphylococcus sp</i> | 0 | 0 | 1 | 0 | 0 | 1 | 2.6 |
| 12 | <i>Streptococcus viridans</i> | 1 | 0 | 0 | 0 | 0 | 1 | 2.6 |
| 13 | <i>Streptococcus anginosus</i> | 1 | 0 | 0 | 0 | 0 | 1 | 2.6 |
| 14 | <i>Streptococcus salivarius</i> | 0 | 1 | 0 | 0 | 0 | 1 | 2.6 |
| 15 | <i>Micrococcus lylae</i> | 0 | 1 | 0 | 0 | 0 | 1 | 2.6 |
| Total | | 14 | 11 | 10 | 3 | 1 | 39 | 100 |

Among Gram-positive bacteria, *Staphylococcus aureus* was the predominant pathogen and represented the most clinically significant Gram-positive isolate in this study. This organism appeared across multiple specimen types—most notably blood—highlighting its importance as a cause of bloodstream and invasive infections. Several coagulase-negative *Staphylococcus* species were also

identified, but in much smaller proportions, suggesting that their contribution was comparatively minor. Overall, the Gram-positive profile indicates that *S. aureus* remains the key Gram-positive pathogen of concern, particularly in samples associated with systemic or device-related infections.

Results of Antibiotic Resistance Testing

Gram-Negative Bacteria

The resistance profile of Gram-negative bacteria in this study shows a consistently high level of resistance to most β -lactam antibiotics, indicating substantial therapeutic limitations. *Acinetobacter baumannii* and *Pseudomonas aeruginosa* demonstrated the most concerning multidrug-resistant patterns, aligning with their classification as WHO critical-priority pathogens. *Klebsiella pneumoniae* and *Escherichia coli* also exhibited considerable resistance to commonly used antibiotics, underscoring the burden of extended-spectrum β -lactamase (ESBL)-related resistance in this setting.

Despite this overall trend, certain antibiotics—particularly amikacin, carbapenems, and piperacillin-tazobactam—still showed reliable activity against several Gram-negative isolates, making them potential empiric options when clinically indicated. These findings highlight a high antimicrobial resistance burden among Gram-negative organisms, with a narrow selection of effective agents remaining.

Table 3. Antibiotic Resistance Test Results for Gram-Negative Bacteria

| No. | Pattern | Gram-Negative Bacteria | | | | | | | | | |
|-----|----------------------------|------------------------|---------------------|----------------|----------------------|---------------------|----------------------|----------------------|---------------------|----------------------|-----|
| | | <i>A. baumannii</i> | <i>E. aerogenes</i> | <i>E. coli</i> | <i>K. pneumoniae</i> | <i>P. mirabilis</i> | <i>P. aeruginosa</i> | <i>S. marcescens</i> | <i>S. odorifera</i> | <i>S. plymuthica</i> | |
| 1. | Ampicillin Sulbactam (SAM) | R | 0 | 3/3 | 50% | 40% | 0 | 2/2 | 1/1 | 1/1 | 0 |
| | | I | ½ | 0 | 0 | 30% | 1/3 | 0 | 0 | 0 | 0 |
| | | S | ½ | 0 | 50% | 30% | 2/3 | 0 | 0 | 0 | 1/1 |
| | | n | 2 | 3 | 6 | 10 | 3 | 2 | 1 | 1 | 1 |
| 2. | Amikacin (AK) | R | 1/3 | 0 | 0 | 10% | 0 | ½ | 0 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 2/3 | 3/3 | 100% | 90% | 3/3 | ½ | 1/1 | 0 | 1/1 |
| | | n | 3 | 3 | 6 | 10 | 3 | 2 | 1 | 1 | 1 |
| 3. | Ampicillin (AMP) | R | 3/3 | 3/3 | 100% | 100% | 2/3 | 1/1 | 1/1 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 0 | 0 | 0 | 0 | 1/3 | 0 | 0 | 0 | 1/1 |
| | | n | 3 | 3 | 6 | 10 | 3 | 1 | 1 | 1 | 1 |
| 4. | Aztreonam (ATM) | R | 3/3 | 3/3 | 67% | 60% | 0 | 2/2 | 0 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 0 | 0 | 33% | 40% | 2/2 | 0 | 1/1 | 0 | 1/1 |
| | | n | 3 | 3 | 6 | 10 | 2 | 2 | 1 | 1 | 1 |
| 5. | Amoxicillin (AMX) | R | 3/3 | N/A | N/A | N/A | N/A | 1/1 | N/A | N/A | N/A |
| | | I | 0 | N/A | N/A | N/A | N/A | 0 | N/A | N/A | N/A |
| | | S | 0 | N/A | N/A | N/A | N/A | 0 | N/A | N/A | N/A |
| | | n | 3 | N/A | N/A | N/A | N/A | 1 | N/A | N/A | N/A |
| 6. | Ciprofloxacin (CIP) | R | ½ | 1/3 | 5/5 | 50% | 2/3 | 0 | 0 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 10% | 0 | 2/2 | 0 | 0 | 0 |
| | | S | ½ | 2/3 | 0 | 40% | 1/3 | 0 | 1/1 | 0 | 1/1 |
| | | n | 2 | 3 | 5 | 10 | 3 | 2 | 1 | 1 | 1 |
| 7. | Cefazolin (KZ) | R | 3/3 | 3/3 | 83% | 50% | 1/3 | N/A | 1/1 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 1/3 | N/A | 0 | 0 | 0 |
| | | S | 0 | 0 | 17% | 50% | 1/3 | N/A | 0 | 0 | 1/1 |
| | | n | 3 | 3 | 6 | 10 | 3 | N/A | 1 | 1 | 1 |
| 8. | Ceftriaxone (CRO) | R | 2/3 | 3/3 | 67% | 60% | 1/3 | 2/2 | 1/1 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 1/3 | 0 | 33% | 40% | 2/3 | 0 | 0 | 0 | 1/1 |
| | | n | 3 | 3 | 6 | 10 | 3 | 2 | 1 | 1 | 1 |
| 9. | Cefoxitin (FOX) | R | 3/3 | 3/3 | 17% | 10% | 0 | N/A | 1/1 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | N/A | 0 | 0 | 0 |
| | | S | 0 | 0 | 83% | 90% | 3/3 | N/A | 0 | 0 | 1/1 |
| | | n | 3 | 3 | 6 | 10 | 3 | N/A | 1 | 1 | 1 |
| 10. | Cefepime (FEP) | R | 3/3 | 2/3 | 33% | 50% | 1/3 | 0 | 0 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 10% | 0 | ½ | 0 | 0 | 0 |
| | | S | 0 | 1/3 | 67% | 40% | 2/3 | ½ | 1/1 | 0 | 1/1 |
| | | n | 3 | 3 | 6 | 10 | 3 | 2 | 1 | 1 | 1 |

| No. | Pattern | Gram-Negative Bacteria | | | | | | | | | |
|-----|-------------------------------------|------------------------|---------------------|----------------|----------------------|---------------------|----------------------|----------------------|---------------------|----------------------|-----|
| | | <i>A. baumannii</i> | <i>E. aerogenes</i> | <i>E. coli</i> | <i>K. pneumoniae</i> | <i>P. mirabilis</i> | <i>P. aeruginosa</i> | <i>S. marcescens</i> | <i>S. odorifera</i> | <i>S. plymuthica</i> | |
| 11. | Cefixime (CFM) | R | 1/1 | 1/1 | ¾ | 71% | 0 | 1/1 | 1/1 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 0 | 0 | ¼ | 29% | 1/1 | 0 | 0 | 0 | 1/1 |
| 12. | Cefuroxime (CXM) | n | 1 | 1 | 4 | 7 | 1 | 1 | 1 | 1 | 1 |
| | | R | 3/3 | 2/2 | 33% | 60% | 1/3 | N/A | 1/1 | 1/1 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | N/A | 0 | 0 | 0 |
| 13. | Cefoperazone/Sulbactam (SCF) | S | 0 | 0 | 67% | 40% | 2/3 | N/A | 0 | 0 | 1/1 |
| | | n | 3 | 2 | 6 | 10 | 3 | N/A | 1 | 1 | 1 |
| | | R | 0 | 0 | 0 | 10% | 1/3 | ½ | 0 | 1/1 | 0 |
| 14. | Ceftazidime (CAZ) | I | 0 | 0 | 0 | 0 | 0 | 0 | 1/1 | N/A | 0 |
| | | S | 2/3 | 0 | 50% | 44% | 2/3 | ½ | 0 | N/A | 1/1 |
| | | n | 3 | 3 | 6 | 9 | 3 | 2 | 1 | N/A | 1 |
| 15. | Cefotaxime (CTX) | R | 1/3 | 3/3 | 50% | 56% | 1/3 | ½ | 0 | N/A | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 1/1 | N/A | 0 |
| | | S | 0 | 0 | ¼ | 38% | 2/3 | 0 | 0 | N/A | 1/1 |
| 16. | Ertapenem (ETP) | n | 1 | 3 | 4 | 8 | 3 | 2 | 1 | N/A | 1 |
| | | R | 3/3 | N/A | N/A | N/A | N/A | 2/2 | N/A | N/A | N/A |
| | | I | 0 | N/A | N/A | N/A | N/A | 0 | N/A | N/A | N/A |
| 17. | Gentamicin (GN) | S | 0 | 0 | 0 | 0 | 0 | 0 | 0 | N/A | 0 |
| | | n | 3 | N/A | N/A | N/A | N/A | 2 | N/A | N/A | N/A |
| | | R | 2/2 | 2/3 | 17% | 40% | 1/3 | ½ | 1/1 | 1/1 | 0 |
| 18. | Meropenem (MEM) | I | 0 | 0 | 0 | 0 | 1/3 | 0 | 0 | 0 | 0 |
| | | S | 0 | 1/3 | 83% | 60% | 1/3 | ½ | 0 | 0 | 1/1 |
| | | n | 2 | 3 | 6 | 10 | 3 | 2 | 1 | 1 | 1 |
| 19. | Trimethoprim-Sulfamethoxazole (OXT) | R | 1/3 | 0 | 0 | 10% | 0 | ½ | 0 | 0 | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 2/3 | 3/3 | 3/3 | 90% | 3/3 | ½ | 1/1 | 1/1 | 1/1 |
| 20. | Tazobactam/Piperacillin (TZP) | n | 3 | 3 | 3 | 10 | 3 | 2 | 1 | 1 | 1 |
| | | R | 2/3 | 0 | 67% | 38% | 3/3 | 2/2 | 0 | N/A | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | N/A | 0 |
| 21. | Tigecycline (TGC) | S | 1/3 | 2/2 | 33% | 62% | 0 | 0 | 1/1 | N/A | 1/1 |
| | | n | 3 | 2 | 6 | 8 | 3 | 2 | 1 | N/A | 1 |
| | | R | 0 | 0 | 0 | 30% | 0 | ½ | 0 | 1/1 | 0 |
| 20. | Tazobactam/Piperacillin (TZP) | I | ½ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | ½ | 2/2 | 100% | 70% | 3/3 | ½ | 1/1 | 0 | 1/1 |
| | | n | 2 | 2 | 6 | 10 | 3 | 2 | 1 | 1 | 1 |
| 21. | Tigecycline (TGC) | R | 0 | 0 | 0 | 25% | 3/3 | 2/2 | 1/1 | 0 | 0 |
| | | I | 0 | ½ | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 3/3 | ½ | 100% | 75% | 0 | 0 | 0 | 1/1 | 1/1 |
| n | 3 | 2 | 6 | 8 | 3 | 2 | 1 | 1 | 1 | | |

R: resistant, I: intermediate, S: susceptible, N/A: not available, n: sample size

Gram-Positive Bacteria

The resistance profile of Gram-positive bacteria in this study is marked by widespread β-lactam resistance, with *Staphylococcus aureus* and coagulase-negative *Staphylococcus* species showing consistently poor susceptibility to penicillins and cephalosporins. This pattern highlights the clinical importance of methicillin resistance among local isolates. Despite this, several last-line agents—particularly vancomycin, linezolid, and gentamicin—remained active against most Gram-positive species, indicating that these antibiotics continue to be reliable options for severe infections. Overall, the data suggest that Gram-positive pathogens in this setting exhibit high β-lactam resistance but retain susceptibility to key anti-MRSA agents, emphasizing the need for careful antibiotic selection, especially in suspected staphylococcal infections.

Table 4. Antibiotic Resistance Test Results for Gram-Positive Bacteria

| No. | Antibiotic | Pattern | Gram-Positive Bacteria | | | | | | | | |
|-----|-------------------------------------|---------|------------------------|-----------------------|-----------------------|------------------|------------------|-------------------|---------------------|----------------------|--------------------|
| | | | <i>S. aureus</i> | <i>S. epidermidis</i> | <i>S. intermedius</i> | <i>S. lentus</i> | <i>S. sciuri</i> | <i>S. xylosum</i> | <i>S. arginosus</i> | <i>S. salivarius</i> | <i>S. viridans</i> |
| 1. | Penicillin (PEN) | R | 27% | 2/3 | 3/3 | ½ | 2/2 | ¾ | 1/1 | 1/1 | 1/1 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 73% | 1/3 | 0 | ½ | 0 | ¼ | 0 | 0 | 0 |
| 2. | Ampicillin (AMP) | n | 15 | 3 | 3 | 2 | 2 | 4 | 1 | 1 | 1 |
| | | R | 3/3 | 0 | 1/1 | 1/1 | N/A | 2/3 | 1/1 | 1/1 | N/A |
| | | I | 0 | 2/2 | 0 | 0 | N/A | 0 | 0 | 0 | N/A |
| 3. | Ciprofloxacin (CIP) | S | 0 | 0 | 0 | 0 | N/A | 1/3 | 0 | 0 | N/A |
| | | n | 3 | 2 | 1 | 1 | N/A | 3 | 1 | 1 | N/A |
| | | R | 64% | 0 | 2/3 | 1/1 | 1/1 | ¾ | 0 | 1/1 | 1/1 |
| 4. | Ceftriaxone (CRO) | I | 7% | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 29% | 1/1 | 1/3 | 0 | 0 | ¼ | 1/1 | 0 | 0 |
| | | n | 14 | 3 | 3 | 1 | 1 | 4 | 1 | 1 | 1 |
| 5. | Cefoxitin (FOX) | R | 54% | 3/3 | 3/3 | 2/2 | 2/2 | ¾ | 1/1 | 1/1 | 1/1 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 46% | 0 | 0 | 0 | 0 | ¼ | 0 | 0 | 0 |
| 6. | Cefoxitin Screen (FOX-S) | n | 13 | 3 | 3 | 2 | 2 | 4 | 1 | 1 | 1 |
| | | R | 47% | 2/3 | 2/3 | 2/2 | 2/2 | 2/3 | N/A | N/A | 0 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | N/A | N/A | 0 |
| 7. | Cefepime (FEP) | S | 53% | 1/3 | 1/3 | 0 | 0 | 1/3 | N/A | N/A | 1/1 |
| | | n | 15 | 3 | 3 | 2 | 2 | 3 | N/A | N/A | 1 |
| | | R | 4/5 | N/A | N/A | 2/2 | 1/1 | 3/3 | N/A | N/A | N/A |
| 8. | Cefixime (CFM) | I | 0 | N/A | N/A | 0 | 0 | 0 | N/A | N/A | N/A |
| | | S | 1/5 | N/A | N/A | 0 | 0 | 0 | N/A | N/A | N/A |
| | | n | 5 | N/A | N/A | 2 | 1 | 3 | N/A | N/A | N/A |
| 9. | Cefuroxime (CXM) | R | 54% | 0 | 2/3 | 2/2 | 2/2 | 2/4 | 0 | 100 | 100 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | S | 46% | 1/1 | 1/3 | 0 | 0 | 2/4 | 100 | 0 | 0 |
| 10. | Ceftazidime (CAZ) | n | 13 | 1 | 3 | 2 | 2 | 4 | 1 | 1 | 1 |
| | | R | 100% | 1/1 | 3/3 | 2/2 | 2/2 | 2/2 | N/A | N/A | 1/1 |
| | | I | 0 | 0 | 0 | 0 | 0 | 0 | N/A | N/A | 0 |
| 11. | Cefotaxime (CTX) | S | 0 | 0 | 0 | 0 | 0 | 0 | N/A | N/A | 0 |
| | | n | 12 | 1 | 3 | 2 | 2 | 2 | N/A | N/A | 1 |
| | | R | 46% | 2/3 | 1/3 | 2/2 | 2/2 | 2/3 | N/A | N/A | 1/1 |
| 12. | Gentamicin (GN) | I | 0 | 0 | 0 | 0 | 0 | 0 | N/A | N/A | 0 |
| | | S | 54% | 1/3 | 2/3 | 0 | 0 | 1/3 | N/A | N/A | 0 |
| | | n | 13 | 3 | 3 | 2 | 2 | 3 | N/A | N/A | 1 |
| 13. | Linezolid (LZD) | R | 4/9 | N/A | ½ | 1/1 | 2/2 | N/A | N/A | N/A | 1/1 |
| | | I | 1/9 | N/A | 0 | 0 | 0 | N/A | N/A | N/A | 0 |
| | | S | 4/9 | N/A | ½ | 0 | 0 | N/A | N/A | N/A | 0 |
| 14. | Meropenem (MEM) | n | 9 | N/A | 2 | 1 | 2 | N/A | N/A | N/A | 1 |
| | | R | 30% | 0 | ½ | 1/1 | 2/2 | N/A | N/A | N/A | 1/1 |
| | | I | 0 | 0 | 0 | 0 | 0 | N/A | N/A | N/A | 0 |
| 15. | Trimethoprim-Sulfamethoxazole (OXT) | S | 70% | 1/1 | ½ | 0 | 0 | N/A | N/A | N/A | 0 |
| | | n | 10 | 1 | 2 | 1 | 2 | N/A | N/A | N/A | 1 |
| | | R | 27% | 0 | 1/3 | ½ | ½ | 0 | 0 | 1/1 | 0 |
| 16. | Vancomycin (VAN) | I | 6% | 0 | 0 | 0 | ½ | 0 | 0 | 0 | 0 |
| | | S | 67% | 1/3 | 3/3 | ½ | ½ | 4/4 | 1/1 | 1/1 | 1/1 |
| | | n | 15 | 3 | 3 | 2 | 2 | 4 | 1 | 1 | 1 |

R: resistant, I: intermediate, S: susceptible, N/A: not available, n: sample size

DISCUSSION

Characteristics Based on Sample Types

The distribution of specimen types submitted for culture indicates that most suspected infections during the study period were bloodstream, soft-tissue, and respiratory infections. Blood cultures represented the largest proportion of specimens, followed by pus and sputum, reflecting the substantial clinical burden posed by these infection types in the hospital setting. This finding is consistent with the study by Hidayat (2016), which also demonstrated that blood specimens dominated the submitted cultures in the ICU and Perinatology units of Dr. H. Abdul Moeloek Hospital. The predominance of blood samples in both studies highlights the significant incidence of suspected bloodstream infections in critically ill patients, where sepsis and septic shock are common clinical presentations requiring rapid etiologic confirmation and targeted therapy [14].

Differences in specimen distribution across studies are largely influenced by variations in patient characteristics, clinical case mix, and ward populations. In our setting, blood, pus, and sputum were the most frequently processed specimens, reflecting a higher burden of suspected bloodstream, soft-tissue, and respiratory infections in the hospital during the study period. Meanwhile, studies from Makassar and Padang showed different dominant specimen types because their patient populations and clinical profiles differed—ICU settings with higher rates of ventilator-associated pneumonia may yield more sputum samples, whereas units managing more systemic infections tend to produce a higher proportion of blood cultures. These differences highlight how local epidemiology, hospital services, and ward-specific infection patterns shape the types of specimens submitted for culture [15, 16].

The differences in the predominant sample types for culture isolates across regions may also be attributed to various factors, including differences in patient profiles, types of healthcare services provided by hospitals, prevailing policies and protocols, the capacity of microbiology laboratories, the spectrum of endemic diseases and local epidemiology, as well as economic factors [5, 17, 18].

Characteristics Based on Bacterial Types

The predominance of *Klebsiella pneumoniae*, *Escherichia coli*, and *Acinetobacter* species in this study reflects their well-established role as leading causes of healthcare-associated infections, particularly in settings with frequent invasive procedures, antibiotic exposure, and prolonged hospitalization. These organisms are common colonizers of the human gastrointestinal tract (*E. coli* and *K. pneumoniae*) and hospital environments (*Acinetobacter*), which facilitates their transmission and increases the likelihood of infection in vulnerable patients. Their ability to acquire and express multiple resistance mechanisms also contributes to their higher detection rates in clinical specimens [19].

Similarly, the dominance of *Staphylococcus aureus*, *Staphylococcus xylosum*, and *Staphylococcus epidermidis* among Gram-positive isolates can be attributed to their presence as common components of human skin and mucosal flora. These organisms frequently cause infections associated with medical devices, bloodstream invasions, and soft-tissue involvement, making them prevalent in both inpatient and outpatient settings [19].

Additionally, Darwaningrum (2021) conducted a study on bacterial patterns and antibiotic resistance at Dr. Dradjat Prawiranegara Regional Hospital, Serang, in 2020. Out of 102 isolates, the most common bacteria were *Escherichia coli* with 41 isolates (40.1%), followed by Gram-positive *Diplococcus* with 18 isolates (17.6%), and *Staphylococcus aureus* with 11 isolates (10.7%). These studies consistently show that the percentage of Gram-negative bacteria is higher than that of Gram-positive bacteria [19]. Gram-positive bacteria were the most common cause of nosocomial infections before the widespread use of antibiotics in the 1940s. However, after antibiotics became widely used, the causative agents of infections changed, and Gram-positive bacteria became less frequently isolated [20].

Results of Antibiotic Resistance Testing on Gram-Negative Bacteria

The predominance of *Klebsiella pneumoniae*, *Escherichia coli*, and *Acinetobacter baumannii* in this study can be explained by their strong intrinsic and acquired resistance mechanisms. Enterobacterales such as *K. pneumoniae* and *E. coli* frequently harbor extended-spectrum β -lactamases (ESBL), enabling them to

hydrolyze penicillins and third-generation cephalosporins. These organisms also possess efficient efflux systems, including the *acrAB*–*tolC* pump, which enhances resistance to multiple classes of antibiotics. In addition, plasmid-mediated virulence factors such as *rmpA* contribute to their survival and persistence in clinical settings by increasing capsule formation and hypervirulence. Together, these factors make *K. pneumoniae* and *E. coli* among the most commonly isolated pathogens in hospital-based studies [21].

Acinetobacter baumannii exhibited high resistance due to its ability to produce OXA-type β -lactamases. The *bla*OXA-23-like carbapenemase is particularly widespread in Indonesia and is a major mechanism underlying carbapenem-resistant *A. baumannii* (CRAB) [22]. This gene has been detected in more than 90% of CRAB isolates in studies from Indonesia, highlighting its dominant role in resistance [23]. Beyond carbapenemase production, *A. baumannii* also demonstrates resistance through porin modification, strong biofilm formation, and prolonged survival on environmental surfaces, which collectively make this pathogen more resistant than many other Gram-negative organisms [22].

Differences in resistance levels among Gram-negative species likely reflect variations in their genetic resistance determinants. Enterobacterales and *A. baumannii* commonly carry ESBLs, carbapenemases, and efflux pump genes that confer broad-spectrum resistance [21, 22, 23]. In contrast, organisms such as *Proteus mirabilis* and *Serratia plymuthica* possess fewer high-potency resistance mechanisms or have limited exposure to broad-spectrum antibiotics in clinical practice, which may explain their comparatively lower resistance profiles observed in both local and international studies [21, 22].

Overall, the predominance and resistance patterns observed in this study reflect a combination of intrinsic bacterial traits, horizontal gene transfer, and selective pressure from antibiotic use in clinical settings [19, 21, 22, 23]. These mechanisms explain why certain pathogens, particularly ESBL-producing Enterobacterales and carbapenem-resistant *A. baumannii*, demonstrate higher resistance levels compared to other organisms [21, 22, 23].

Results of Antibiotic Resistance Testing on Gram-Positive Bacteria

The Gram-positive isolates in this study showed high resistance primarily to penicillins and cephalosporins. This pattern is expected because many Gram-positive pathogens, especially *Staphylococcus aureus* and coagulase-negative staphylococci (CoNS), produce β -lactamase enzymes or express altered penicillin-binding proteins (such as PBP2a), which render β -lactam antibiotics ineffective. These mechanisms explain the high resistance levels observed in *S. aureus*, *S. xylosus*, and *S. epidermidis*, which appeared as the predominant Gram-positive species in our setting [23].

The higher resistance observed in *S. aureus*—particularly methicillin-resistant *S. aureus* (MRSA)—is largely driven by the *mecA* gene, which encodes PBP2a. This altered target protein reduces β -lactam binding affinity, leading to broad resistance to the penicillin and cephalosporin classes. Coagulase-negative staphylococci, including *S. epidermidis* and *S. xylosus*, frequently harbor similar mechanisms and have a strong propensity for biofilm formation on medical devices, further increasing tolerance to antimicrobial exposure. These features explain why Gram-positive staphylococcal species tend to be more resistant than streptococci or other Gram-positive bacteria [23].

Linezolid remained highly effective in this study. As an oxazolidinone, linezolid inhibits bacterial protein synthesis by binding to the 23S rRNA of the 50S ribosomal subunit, preventing initiation of translation. Because linezolid targets ribosomal structures rather than the cell wall, its activity is unaffected by β -lactamases or PBP alterations—mechanisms commonly used by resistant Gram-positive bacteria. This explains why linezolid maintains strong activity against MRSA, VRE, and multiple staphylococcal and streptococcal species [24].

Gentamicin remained an additional effective option for several Gram-positive isolates. Gentamicin acts by binding to the 30S ribosomal subunit, disrupting protein synthesis and damaging the cell membrane. Resistance occurs when *S. aureus* reduces ribosomal binding or produces aminoglycoside-modifying enzymes; however, many isolates still lack these mechanisms, allowing gentamicin to retain activity. Alhumaid et al. also reported high gentamicin susceptibility among *S. aureus*, including MRSA, consistent with our findings [25].

Vancomycin remained effective in this study because its mechanism—blocking cell wall synthesis by

binding to D-Ala-D-Ala precursors—is not affected by β -lactamase production or PBP2a alterations. This makes vancomycin a reliable therapy for resistant staphylococcal infections. Evidence from a long-term study in Iran demonstrated declining vancomycin resistance over the past decade, supporting its sustained value as a treatment for severe *Staphylococcus* infections [26].

CONCLUSION

This study demonstrates a high burden of antimicrobial resistance among clinical isolates at Advent Hospital Bandar Lampung, particularly involving ESBL-producing Enterobacterales and carbapenem-resistant *Acinetobacter baumannii*. The consistently poor activity of ampicillin and most third-generation cephalosporins against Gram-negative organisms indicates that these agents are no longer suitable as empirical options for suspected Gram-negative infections in this setting. In contrast, meropenem, amikacin, cefoperazone-sulbactam, and piperacillin-tazobactam retained good activity and may be considered as potential empirical choices when clinically appropriate and guided by stewardship oversight. For Gram-positive pathogens, *Staphylococcus aureus* and coagulase-negative staphylococci demonstrated high resistance to β -lactams, supporting the continued use of vancomycin, linezolid, or gentamicin for severe infections pending culture results. These findings underscore the need to update empirical therapy guidelines, strengthen antimicrobial stewardship interventions, and enhance infection prevention and control strategies within the hospital. Continuous surveillance is essential to track resistance trends and guide rational antibiotic use.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

REFERENCES

- [1] De Angelis, G., Murthy, A., Beyersmann, J., Harbarth, S., "Estimating the impact of healthcare-associated infections on length of stay and costs," *Clinical Microbiology and Infection*, p. 1729–35, 2010; 16.
- [2] Adefisoye, M.A., Olaniran, A.O., "Antimicrobial resistance expansion in pathogens: A review of current mitigation strategies and advances towards innovative therapy," *JAC Antimicrob Resist*, vol. 5, no. 6, p. dlad127, 2023 Dec 11.
- [3] Pan, Z., Fan, L., Zhong, Y., Guo, J., Dong, X., Xu, X., "Quantitative proteomics reveals reduction in central carbon and energy metabolisms contributes to gentamicin resistance in *Staphylococcus aureus*," *Journal of Proteomics*, vol. 277, p. 104849, 2023.
- [4] Lin, S.Y., Lu, P.L., Wu, T.S., Shie, S. Sen, Chang, F.Y., "Correlation Between Cefoperazone/Sulbactam MIC Values and Clinical Outcomes of *Escherichia coli* Bacteremia," *Infect Dis Ther*, vol. 11, p. 1853–67, 2022.
- [5] Truong, W.R., Hidayat, L., Bolaris, M.A., Nguyen, L., Yamaki, J., "The antibiogram: Key considerations for its development and utilization," *JAC Antimicrob Resist*, vol. 3, no. 2, p. dlab060, 2021 May 25.
- [6] Gach, M.W., Lazarus, G., Simadibrata, D.M., Sinto, R., Saharman, Y.R., Limato, R., Nelwan, E.J., van Doorn, H.R., Karuniawati, A., Hamers, R.L., "Antimicrobial resistance among common bacterial

- pathogens in Indonesia: a systematic review," *The Lancet Regional Health - Southeast Asia* 26, vol. 26, p. 100414, 2024.
- [7] World Health Organization., "WHO Bacterial Priority Pathogens List 2024: Bacterial pathogens of public health importance to guide research, development and strategies to prevent and control antimicrobial resistance," 2024.
- [8] Ministry of Health of the Republic of Indonesia., "Regulation of the Minister of Health No. 8 of 2015 on Antimicrobial Resistance Control Program in Hospitals," 02 March 2015. [Online]. Available: <https://peraturan.bpk.go.id/Details/114886/permenkes-no-8-tahun-2015>. [Accessed 01 September 2025].
- [9] Ministry of Health of the Republic of Indonesia., "Regulation of the Minister of Health No. 27 of 2017 on Guidelines for Infection Prevention and Control in Health Care Facilities," 19 June 2017. [Online]. Available: <https://peraturan.bpk.go.id/Details/112075/permenkes-no-27-tahun-2017>. [Accessed 01 September 2025].
- [10] Coordinating Ministry for Human Development and Cultural Affairs of the Republic of Indonesia., "Regulation No. 7 of 2021 on the National Action Plan for the Control of Antimicrobial Resistance," 19 October 2021. [Online]. Available: <https://peraturan.bpk.go.id/Details/255302/permenko-pmk-no-7-tahun-2021>. [Accessed 01 September 2025].
- [11] Clinical and Laboratory Standards Institute (CLSI)., M100 Performance Standards for Antimicrobial Susceptibility Testing 30th edition. Wayne, PA 19087 USA., Wayne, Pennsylvania, USA: Clinical and Laboratory Standards Institute (CLSI), 2020.
- [12] World Health Organization (WHO). , "Global Antimicrobial Resistance and Use Surveillance System (GLASS) Manual for Early Implementation," WHO, Geneva, 2022.
- [13] Centers for Disease Control and Prevention (CDC). , "Antibiotic Resistance Laboratory Network (ARLN) Surveillance and Reporting Guidelines," CDC, Atlanta, 2021.
- [14] Hidayat., "Analisis Pola Kuman dan Pola Resistensi Antibiotik di Ruang ICU dan Ruang Perinatologi Rumah Sakit Umum Daerah dr. H. Abdul Moeloek Provinsi Lampung Tahun 2013," *Jurnal Medika Malahayati*, vol. 3, no. 1, p. 1–14, 2016.
- [15] Handayani, I., Abdul Kadir, N., "Patterns of germs before, during and after the COVID-19 pandemic in Intensive Care Unit (ICU) patients at Dr. Wahidin Sudirohusodo, Makassar, Indonesia," *Journal of Biomedical Science (IJBS)*, vol. 17, p. 262–68, 2023.
- [16] Putra, M.F.R., Bahar, E., Gustia, R., Linosefa, Russilawati, Julizar., "Pola Bakteri dan Sensitivitas Antibiotik Pada Hasil Kultur Pasien di Ruang Intensive Care Unit RSUP Dr. M. Djamil Padang Tahun 2020," *Sentri: Jurnal Riset Ilmiah*, vol. 3, no. 10, p. 4737–48, 2024.
- [17] Budayanti, N.S., Aisyah, D.N., Fatmawati, N.N.D., Tarini, N.M.A., Kozlakidis, Z., Adisasmito, W., "Identification and Distribution of Pathogens in a Major Tertiary Hospital of Indonesia," *Frontier in Public Health*, vol. 7, p. 395, 2020 Jan 31.
- [18] Lokida, D., Farida, H., Triasih, R., Mardian, Y., Kosasih, H., Naysilla, A.M., et al., "Epidemiology of community-acquired pneumonia among hospitalised children in Indonesia: A multicentre, prospective study," *BMJ Open*, vol. 12, p. e057957, 2022.
- [19] Darwaningrum, R., "Pola Kuman dan Pola Resistensi Terhadap Antibiotik Pada Spesimen Pus di RSUD dr. Dradjat Prawiranegara Serang Tahun 2020," 2021. [Online]. Available: <https://perpustakaan.poltekkesbanten.ac.id/repository/index.php?p=fstream-pdf&fid=502&bid=1779>. [Accessed 01 September 2025].
- [20] Ladyani, F., Zahra, M., "Analisis Pola Kuman dan Pola Resistensi Pada Hasil Pemeriksaan Kultur Resistensi di Laboratorium Patologi Klinik Rumah Sakit dr. H. Abdoel Moeloek Provinsi Lampung Periode Januari-Juli 2016," *Jurnal Ilmu Kedokteran Dan Kesehatan.*, vol. 5, no. 2, pp. 77-88, 2018.

- [21] Mardhia, M., Liana, D.F., Mahyarudin, M., Ih, H., "The first report of antibiotic resistance and virulence factor profiles in multidrug-resistant clinical isolates of *Klebsiella pneumoniae* from Pontianak, Indonesia," *Osong Public Health Res Perspect*, vol. 16, pp. 160-68, 2025.
- [22] Homenta, H., Julyadharna, J., Susianti, H., Noorhamdani, N., Santosaningsih, D., "Molecular Epidemiology of Clinical Carbapenem-Resistant *Acinetobacter baumannii-calcoaceticus* complex Isolates in Tertiary Care Hospitals in Java and Sulawesi Islands, Indonesia," *Trop. Med. Infect. Dis.*, vol. 7, no. 277, 2022.
- [23] Saharman, Y.R., Karuniawati, A., Sedono, R., Aditjaningsih, D., Sudarmono, P., Goessens, W.H.F., "Endemic carbapenem-nonsusceptible *Acinetobacter baumannii-calcoaceticus* complex in intensive care units of the national referral hospital in Jakarta, Indonesia," *Antimicrobial Resistance and Infection Control*, vol. 7, no. 5, 2018.
- [24] Helmy, A.K., Sidkey, N.M., El-Badawy, R.E., Hegazi, A.G., "Emergence of microbial infections in some hospitals of Cairo, Egypt: studying their corresponding antimicrobial resistance profiles.," *BMC Infectious Diseases*, vol. 23, no. 424, 2023.
- [25] Peykov, S., Kirov, B., Strateva, T., "Linezolid in the Focus of Antimicrobial Resistance of *Enterococcus* Species: A Global Overview of Genomic Studies," *Int. J. Mol. Sci*, vol. 26, no. 8207, 2025.
- [26] Alhumaid, S., Mutair, AA., Alawi, ZA., Alzahrani, AJ., Tobaiqy, M., Alresasi, AM., "Antimicrobial susceptibility of gram-positive and gram-negative bacteria: a 5-year retrospective analysis at a multi-hospital healthcare system in Saudi Arabia," *Annals of Clinical Microbiology and Antimicrobials*, vol. 20(1), no. 1, pp. 20-43, 2021.