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"CLINICAL AND MICROBIOLOGICAL PROFILE OF GRAM-NEGATIVE BACTERIA CAUSING ORTHOPEDIC IMPLANT SURGICAL SITE INFECTIONS"

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ABSTRACT **Background:** Orthopedic implant–associated infections (OIAIs) are difficult to eradicate due to biofilm formation and rising antimicrobial resistance. While Gram-positive cocci are classically implicated, Gram-negative bacilli (GNB) are increasingly reported in surgical site infections (SSIs) involving implants. **Methods:** This prospective observational study (January–December 2024) was conducted in the Departments of Microbiology of a tertiary hospital. Of 511 postoperative specimens from patients with implant-related SSI, 305 were culture-positive and analyzed. Isolates were identified by standard microbiological methods. Antimicrobial susceptibility testing followed CLSI 2024 (Kirby–Bauer); ESBL was confirmed by combined disc testing. Colistin MICs were determined by broth microdilution, with selected isolates tested on VITEK-2. **Results:** Of 305 culture-positive infections, 186 (61.0%) were GNB. Most patients were >50 years (64.9%), with males predominating (60.2%). Specimens were pus (52.2%) and wound swabs (47.8%). The leading GNB were *Klebsiella pneumoniae* (34.4%), *Pseudomonas aeruginosa* (21.0%), *Escherichia coli* (17.7%) and *Acinetobacter baumannii* (13.4%), with *Proteus mirabilis*, *Enterobacter spp.* and *Citrobacter spp.* less frequent. The hip (26.3%), knee (18.8%) and spine (16.1%) were the most common infection sites. Carbapenems showed the best activity (e.g., meropenem/imipenem: *E. coli* 81.8%/69.7%, *Enterobacter* 90%/90%), though resistance was notable in *K. pneumoniae* (~50%) and *A. baumannii* (56–60%). *P. aeruginosa* was highly susceptible to netilmicin (94.9%). Doxycycline showed good activity against *Klebsiella* (60.9%), *E. coli* (63.6%) and *Acinetobacter* (88.0%). Cephalosporins and fluoroquinolones performed poorly, reflecting ESBL prevalence. **Conclusion:** GNB form the majority of OIAIs, led by *K. pneumoniae*, *P. aeruginosa* and *E. coli*, with significant multidrug resistance. Empiric cephalosporins or fluoroquinolones are unreliable; carbapenems remain essential but require careful stewardship. Targeted agents such as netilmicin and minocycline may be valuable adjuncts.

KEYWORDS : *orthopedic implant infection; Gram-negative bacilli; antimicrobial resistance.*

INTRODUCTION

Orthopedic implant infections remain among the most challenging complications of musculoskeletal surgery, leading to implant failure, repeated procedures, prolonged antibiotics and suboptimal functional outcomes. Their persistence is strongly linked to biofilm formation on implant surfaces, which shields bacteria from host defenses and conventional therapy; eradication may require prosthesis removal in refractory cases [1,2].

Although *Staphylococcus aureus* and *Staphylococcus epidermidis* classically dominate implant-associated infections, Gram-negative bacteria (GNB) are increasingly recognized as clinically important pathogens in surgical site infections (SSIs) involving implants. Hospital and regional reports from orthopedic settings document frequent isolation of GNB—notably *Escherichia coli*, *Klebsiella* spp. and *Pseudomonas* spp.—alongside Gram-positives, underscoring a broader microbiological spectrum than traditionally appreciated [1,3,4]. Studies in arthroplasty and fixation devices further show distinctive susceptibility profiles at implant sites, highlighting the need for local surveillance to guide empiric choices [5,6].

The clinical impact of GNB in implant SSIs is amplified by biofilm-mediated tolerance and antimicrobial resistance. Biofilms limit antibiotic penetration, reduce metabolic activity and enable expression of specific resistance determinants, promoting persistent or recurrent infection despite apparently appropriate therapy [7–9]. Concomitantly, orthopedic series from India and elsewhere report rising resistance among GNB, including extended-spectrum β-lactamase (ESBL) phenotypes and reduced susceptibility to key β-lactams and fluoroquinolones, thereby narrowing options and increasing reliance on targeted, culture-directed regimens [1,3,4,5].

Against this backdrop, defining the local burden, species distribution and resistance patterns of Gram-negative pathogens in implant-associated SSIs is essential for rational prophylaxis, optimized empiric therapy and antimicrobial stewardship. The present work focuses specifically on GNB in orthopedic implant infections, integrating organism profiles with resistance trends to inform evidence-based management and improve outcomes.

Objective

To identify the bacteriological spectrum of Gram-negative bacteria causing surgical site infections in patients with orthopedic implants.

METHODOLOGY

Study Design And Setting

This was a prospective observational study carried out in the Departments of Microbiology of a tertiary care hospital over 18 months (1 January 2024 to 31 December 2024). Institutional Ethics Committee approval and informed consent were obtained prior to enrollment.

Study Population And Sample Size

All patients undergoing orthopedic surgeries with implant placement who developed clinical signs of surgical site infection (SSI) were included. During the study period, 511 samples were processed, of which 305 yielded culture-positive results and were analyzed.

Inclusion Criteria:

- Orthopedic implant patients with post-operative clinical signs of infection.
- Patients providing informed written consent.

Exclusion Criteria:

- Refusal to consent.
- Improperly labeled/insufficient specimens or those lacking essential clinical details.
- Samples with compromised diagnostic integrity.

Sample Collection And Transport

Specimens (pus, wound swabs, aspirates, tissue biopsies) were collected aseptically and immediately transported to the microbiology laboratory. Swabs were placed in sterile test tubes, moistened with sterile saline when required and labeled with patient identifiers. Samples were transported promptly to prevent desiccation.

Sample Processing

- **Direct microscopy:** Gram staining was performed for preliminary differentiation.
- **Culture:** Samples were inoculated on Blood Agar and MacConkey Agar, incubated aerobically at 37 °C for 24–48 hours and observed for up to 3 days.
- **Identification:** Isolates were characterized by colony morphology, lactose fermentation, motility and standard

biochemical tests including catalase, oxidase, indole, citrate utilization, urease, triple sugar iron (TSI), methyl red (MR), Voges-Proskauer (VP), nitrate reduction and amino acid decarboxylation (lysine, arginine, ornithine). Specific identification protocols were applied for *Pseudomonas aeruginosa* and *Acinetobacter baumannii*.

Antimicrobial Susceptibility Testing (AST)

AST was performed on all Gram-negative isolates using the Kirby–Bauer disc diffusion method on Mueller–Hinton agar according to CLSI 2024 guidelines. Antibiotic panels included β-lactams, carbapenems, aminoglycosides, fluoroquinolones, tetracyclines, polymyxins and tigecycline.

- **ESBL detection:** Screening with ceftazidime (30 µg) and cefotaxime (30 µg), followed by confirmatory combined disc tests with clavulanic acid.
- **Colistin susceptibility:** Determined by broth microdilution (BMD) using cation-adjusted Mueller–Hinton broth in 96-well microtiter plates. The MIC was recorded as the lowest concentration with no visible growth. *E. coli* ATCC 25922 and *P. aeruginosa* ATCC 27853 were used as controls.
- **Automated AST:** Selected isolates were further tested with the VITEK-2 system for MIC determination, which employs miniaturized broth microdilution and colorimetric growth analysis.

DATA COLLECTION AND ANALYSIS

Demographic and clinical details were recorded. Microbiological and susceptibility data were entered in Microsoft Excel and analyzed using SPSS. Descriptive statistics (frequency, percentage) were applied for organism distribution and resistance patterns.

RESULTS

Out of 305 culture-positive orthopedic implant infections, 186 cases (61.0%) were due to Gram-negative bacilli, while the remaining 119 (39.0%) were Gram-positive organisms. The age distribution showed the highest involvement in the 51–60 years group (46 cases, 24.7%), followed by 61–70 years (40 cases, 21.5%) and >70 years (34 cases, 18.3%). The least affected group was <20 years (5 cases, 2.7%). Out of 186 GNB cases, 112 were males (60.2%) and 74 were females (39.8%). Gram-negative isolates were almost equally distributed between pus and wound swabs. 97 isolates (52.2%) were from pus samples and 89 isolates (47.8%) from wound swabs. Among Gram-negative isolates, *Klebsiella pneumoniae* (64; 34.4%) was the most common, followed by *Pseudomonas aeruginosa* (39; 21.0%) and *Escherichia coli* (33; 17.7%). *Acinetobacter baumannii* accounted for 25 cases (13.4%), while *Proteus mirabilis*, *Enterobacter spp.* and *Citrobacter spp.* were less frequent.

The hip joint was the most common site of Gram-negative infections (49 cases, 26.3%), followed by the knee (35 cases, 18.8%) and spine (30 cases, 16.1%). Upper limb and lower limb long bones (humerus, tibia, femur) together constituted a significant proportion of cases. Among Gram-negative isolates, Klebsiella pneumoniae was the most frequently isolated pathogen (64 cases, 34.4%), followed by Pseudomonas aeruginosa (39 cases, 21.0%) and Escherichia coli (33 cases, 17.7%). Acinetobacter baumannii accounted for 25 cases (13.4%), while Proteus mirabilis (11; 5.9%), Enterobacter spp. (10; 5.4%) and Citrobacter spp. (4; 2.2%) were less common. The hip joint was the predominant site of infection (49 cases, 26.3%), followed by the knee (35 cases, 18.8%), spine (30 cases, 16.1%), humerus (24 cases, 12.9%), tibia (21 cases, 11.3%) and femur (16 cases, 8.6%). Other sites constituted 11 cases (5.9%). (Table 1)

The antibiotic susceptibility pattern of Gram-negative bacilli is presented in Tables 2–4.

Among aminoglycosides, amikacin and gentamicin showed moderate sensitivity in *Klebsiella* (28.1% and 32.8%, respectively), but higher rates in *Pseudomonas* (59.0% and 71.8%) and *Enterobacter* (80.0% for both). *E. coli* demonstrated good sensitivity to both amikacin (54.5%) and gentamicin (57.6%). *Pseudomonas* showed excellent susceptibility to netilmicin (94.9%). Carbapenems exhibited the highest activity overall, with imipenem and meropenem sensitivities ranging from 51.6% to 81.8% across different organisms and particularly high in *Enterobacter* (90%) and *Citrobacter* (75–100%). Among cephalosporins, sensitivity was variable, being generally lower in *Klebsiella* but higher in *E. coli* and *Pseudomonas* (e.g., ceftazidime: 57.6% in *E. coli*, 59.0% in *Pseudomonas*). Tetracyclines showed promising activity, especially doxycycline (60.9% in *Klebsiella*, 63.6% in *E. coli*, 88.0% in *Acinetobacter*). (Table 2)

Intermediate susceptibility was noted in a subset of isolates. Notably, ciprofloxacin exhibited intermediate resistance in *Pseudomonas* (33.3%) and *E. coli* (12.1%). Cephalosporins such as ceftriaxone and ceftazidime also demonstrated intermediate activity in several species (up to 30% in *Enterobacter* and *Citrobacter*). (Table 3)

High resistance rates were observed to many antibiotics. Klebsiella showed high resistance to fluoroquinolones (ciprofloxacin 78.1%) and cephalosporins (ceftriaxone 75.0%, cefotaxime 62.5%). *E. coli* exhibited 78.8% resistance to ampicillin and 51.5% to ciprofloxacin. *Acinetobacter* was highly resistant to piperacillin–tazobactam (64.0%) and fluoroquinolones (52.0%). *Proteus* and *Citrobacter* showed variable but significant resistance across multiple classes. (Table 4)

Table 1: Gram-negative Bacteria In Orthopedic Implant Infections (n = 186)

Category	Subgroup	Frequency (n)	Percentage (%)
Culture Results	Gram-negative bacilli	186	61.0
	Gram-positive organisms	119	39.0
Age Group (years)	<20	5	2.7
	21–30	12	6.5
	31–40	20	10.8
	41–50	29	15.6
	51–60	46	24.7
	61–70	40	21.5
	>70	34	18.3
Gender	Male	112	60.2
	Female	74	39.8
Sample Type	Pus	97	52.2
	Wound Swab	89	47.8
Organism	Klebsiella pneumoniae	64	34.4
	Pseudomonas aeruginosa	39	21.0
	Escherichia coli	33	17.7
	Acinetobacter baumannii	25	13.4
	Proteus mirabilis	11	5.9
	Enterobacter spp.	10	5.4
	Citrobacter spp.	4	2.2
Site of Infection	Hip	49	26.3
	Knee	35	18.8
	Spine	30	16.1
	Humerus	24	12.9
	Tibia	21	11.3
	Femur	16	8.6
	Others	11	5.9

DISCUSSION

In this cohort of 305 culture-positive orthopedic implant infections, Gram-negative bacilli (GNB) comprised a substantial proportion (61.0%), exceeding Gram-positive organisms (39.0%). Nearly two-thirds of culture-positive patients were aged >50 years (64.9%), with the highest burden in the 51–60 year group (24.7%), followed by 61–70 years (21.5%) and >70 years (18.3%). This age gradient mirrors the findings of Borgohain et al. [10], who observed peak infection rates in patients >60 years, attributing risk to immunosenescence and multimorbidity. While some studies describe mid-age peaks (31–50 years) (Lakshminarayana et al. [11]; Kaipa et al. [12]) or a lower mean age (Aditya et al. [13]), the present data reinforce advanced age as a major susceptibility factor—likely driven by greater arthroplasty volume, delayed wound healing, diabetes, renal dysfunction and nutritional deficits. Conversely, younger-age peaks reported by Das et al. [14] (20–29 years) and Perumal et al. [15] (30–39 years) likely reflect local trauma epidemiology and fixation practices.

A mild male predominance was observed (59.0% vs 41.0%; M:F ≈1.44:1). This aligns with the preponderance of males noted across several datasets-often more pronounced than in our series (e.g., Jyoti et

al. 77.5% male; Waliullah et al. [16] ~5:1; Perumal et al. [15] 84.5% male). Explanations include higher exposure to trauma, physically demanding occupations and lifestyle-related risks. Behavioural factors (e.g., smoking, alcohol use) and delayed care-seeking may further elevate risk. At the population level, Borgohain et al. [10] also reported higher infection rates in males (13.96% vs 9.23%), while Aditya et al. [13] documented a 2:1 male-to-female ratio.

GNB were isolated almost equally from pus (52.2%) and wound swabs (47.8%). The slightly higher yield from pus reinforces the superiority of deep aspirates or tissue specimens over superficial swabs, which may underperform in biofilm-associated infections and risk contamination. Prior work similarly favours pus/tissue for culture and susceptibility testing (Lakshminarayana et al. [11]; Sarangi et al. [17]; Perumal et al. [15]). Although some series did not stratify by sample type (Alelign et al. [18]; Kaipa et al. [12]), the consensus remains to prioritise deep samples whenever feasible.

Among GNB (n=186), *Klebsiella pneumoniae* was predominant (34.4%), followed by *Pseudomonas aeruginosa* (21.0%) and *Escherichia coli* (17.7%), with *Acinetobacter baumannii* (13.4%) and smaller proportions of *Proteus*, *Enterobacter* and *Citrobacter*. This distribution overlaps with multiple reports where *K. pneumoniae*, *P. aeruginosa* and *E. coli* consistently rank among leading implant pathogens (Borgohain et al. [10]; Lakshminarayana et al. [11]; Kaipa et al. [12]; Aditya et al. [13]; Das et al. [14]; Perumal et al. [15]; Waliullah et al. [16]; Sarangi et al. [17]; Alelign et al. [18]; Okoro et al. [19]). Differences in order and frequency may reflect referral patterns, antimicrobial exposure and the proportion of arthroplasty versus trauma implants. The prominence of *K. pneumoniae* and *P. aeruginosa* underscores the clinical importance of ESBL production, carbapenem use and biofilm persistence.

The hip (26.3%), knee (18.8%) and spine (16.1%) were the most common sites, followed by long bones (humerus, tibia, femur). This distribution parallels implant placement in weight-bearing joints and major bones, where surgery is complex and foreign-body load favours biofilm formation. Prior studies variably emphasised long bones or joints depending on case mix: femur/tibia predominance (Borgohain et al. [10]; Aditya et al. [13]; Okoro et al. [19]); higher burdens in trauma femur/acetabulum and arthroplasty sites (Waliullah et al. [16]); implant-level associations with nails, plates and K-wires (Kaipa et al. [12]; Perumal et al. [15]); and procedure-linked risk in ORIF/CRIF cohorts (Das et al. [14]). Our hip- and knee-dominant infections align with the rising arthroplasty load in ageing populations.

In terms of antibiotic susceptibility, GNB in this study exhibited widespread multidrug resistance. Carbapenems remained the most reliable class, with *E. coli* (meropenem 81.8%, imipenem 69.7%), *Enterobacter* (90% each) and *Proteus mirabilis* (81.8% and 72.7%) showing the highest activity. However, resistance was pronounced in *K. pneumoniae* (≈50%) and moderate in *A. baumannii* (56–60%). Cephalosporins performed poorly, with ceftriaxone, cefotaxime and cefepime demonstrating low sensitivity in *Klebsiella* and *E. coli*, consistent with ESBL production. Fluoroquinolone efficacy was also limited, with ciprofloxacin sensitivity as low as 17.2% in *Klebsiella* and 24.0% in *Acinetobacter* and only moderate activity in *Pseudomonas* (48.7%).

Aminoglycosides retained moderate but species-specific efficacy. *E. coli* and *Enterobacter* showed 55–80% sensitivity to amikacin and gentamicin, while *Klebsiella* isolates were less responsive. *Pseudomonas aeruginosa* was best treated with netilmicin (94.9%), followed by gentamicin (71.8%) and tobramycin (66.7%). Among β-lactam/β-lactamase inhibitor combinations, piperacillin–tazobactam and amoxicillin–clavulanic acid were largely ineffective against *Klebsiella* and *E. coli* but showed moderate activity against *Enterobacter* and *Proteus*. Tetracyclines offered additional therapeutic value, with doxycycline effective against *Klebsiella* (60.9%), *E. coli* (63.6%) and *Acinetobacter* (88.0%); minocycline was particularly effective in *Acinetobacter* (88.0%).

These findings are consistent with several published reports. Das et al. [14] documented *Klebsiella* as the predominant isolate with high resistance to third- and fourth-generation cephalosporins but preserved carbapenem sensitivity, closely paralleling our results. Waliullah et al. [16] reported carbapenems and aztreonam as the most effective drugs, echoing our observations. Lakshminarayana et al. [11] also described *Klebsiella* resistance to β-lactams with better carbapenem activity, while Perumal et al. [15] noted broad resistance to ampicillin, cephalosporins and cotrimoxazole but better imipenem and piperacillin–tazobactam activity. For *Pseudomonas aeruginosa*, our findings of high carbapenem and aminoglycoside susceptibility but poor cephalosporin and fluoroquinolone activity match the reports of Aditya et al. [13], Sarangi et al. [17] and Alelign et al. [18]. Borgohain et al. [10] and Okoro et al. [19] also highlighted *Pseudomonas* as a frequent isolate with reliable carbapenem and aminoglycoside activity.

Acinetobacter baumannii in this study displayed multidrug resistance but remained sensitive to minocycline, ampicillin–sulbactam and tobramycin, consistent with Das et al. [14], Perumal et al. [15] and Borgohain et al. [10]. *Proteus mirabilis* and *Enterobacter spp.* retained good carbapenem and aminoglycoside susceptibility but were resistant to cephalosporins, in line with Alelign et al. [18], Kaipa et al. [12] and Waliullah et al. [16]. *Citrobacter spp.*, though infrequent, showed poor cephalosporin and fluoroquinolone activity but remained susceptible to carbapenems, supported by Kaipa et al. [12] and Perumal et al. [15].

Overall, the AST profile in our study mirrors published literature, with carbapenems emerging as the cornerstone of therapy but showing concerning resistance trends. Cephalosporins and fluoroquinolones are unreliable empirically, while aminoglycosides and tetracyclines provide adjunct options in selected organisms. These data highlight the importance of culture-directed therapy, robust antimicrobial stewardship and early detection of ESBL- and carbapenemase-producing strains to optimise outcomes in orthopedic implant infections.

CONCLUSION

Gram-negative bacilli accounted for the majority of orthopedic implant infections in this study, with *Klebsiella pneumoniae*, *Pseudomonas aeruginosa* and *Escherichia coli* as the leading pathogens. A high prevalence of multidrug resistance was observed, with poor efficacy of cephalosporins and fluoroquinolones and only partial preservation of carbapenem activity. Aminoglycosides and tetracyclines retained organism-specific value, particularly netilmicin for *Pseudomonas* and minocycline for *Acinetobacter*. These findings underscore the urgent need for culture-guided therapy, strict antimicrobial stewardship and targeted infection-control strategies to improve outcomes in implant-associated infections.

Limitations

While this study provides important insights into the bacteriological spectrum and antimicrobial resistance patterns of orthopedic implant infections, its single-center design may limit generalizability to other regions with different surgical practices and microbial ecology. Biofilm detection methods were not employed, which could have clarified resistance mechanisms and recurrence risk. Furthermore, clinical outcomes such as infection resolution, implant retention or revision were not assessed and infection trends were not stratified by surgical technique (e.g., primary vs revision), limiting the broader clinical applicability of the findings.

Conflict Of Interest: Nil

Funding: Nil

Table 2: Antibiotic Susceptibility Pattern – Sensitive (n/%)

Antibiotic	Klebsiella (n=64)	Pseudomonas (n=39)	E. coli (n=33)	Acinetobacter (n=25)	Enterobacter (n=10)	Proteus (n=11)	Citrobacter (n=4)
Amikacin	18 (28.1%)	23 (59.0%)	18 (54.5%)	9 (36.0%)	8 (80.0%)	8 (72.7%)	2 (50.0%)
Gentamicin	21 (32.8%)	28 (71.8%)	19 (57.6%)	10 (40.0%)	8 (80.0%)	7 (63.6%)	2 (50.0%)
Tobramycin	–	26 (66.7%)	–	17 (68.0%)	–	–	–
Netilmicin	–	37 (94.9%)	–	–	–	–	–
Ciprofloxacin	11 (17.2%)	19 (48.7%)	12 (36.4%)	6 (24.0%)	4 (40.0%)	4 (36.4%)	1 (25.0%)
Ceftriaxone	13 (20.3%)	–	15 (45.5%)	–	2 (20.0%)	5 (45.5%)	1 (25.0%)

Cefotaxime	22 (34.4%)	–	19 (57.6%)	–	–	–	–
Ceftazidime	–	–	19 (57.6%)	14 (56.0%)	3 (30.0%)	5 (45.5%)	1 (25.0%)
Cefepime	26 (40.6%)	–	13 (39.4%)	–	5 (50.0%)	5 (45.5%)	2 (50.0%)
Cefoxitin	21 (32.8%)	–	–	–	–	5 (45.5%)	–
Cefuroxime	20 (31.3%)	–	11 (33.3%)	–	3 (30.0%)	3 (27.3%)	1 (25.0%)
Ceftaroline	–	–	18 (54.5%)	–	2 (50.0%)	–	2 (50.0%)
Aztreonam	–	30 (76.9%)	–	–	–	–	–
Ceftazidime + Avibactam	–	23 (59.0%)	–	–	–	–	–
Imipenem	33 (51.6%)	29 (74.4%)	23 (69.7%)	14 (56.0%)	9 (90.0%)	8 (72.7%)	3 (75.0%)
Meropenem	31 (48.4%)	29 (74.4%)	27 (81.8%)	15 (60.0%)	9 (90.0%)	9 (81.8%)	4 (100.0%)
Ertapenem	27 (42.2%)	–	25 (75.8%)	–	9 (90.0%)	5 (45.5%)	2 (50.0%)
Piperacillin–Tazobactam	19 (29.7%)	26 (66.7%)	7 (21.2%)	5 (20.0%)	7 (70.0%)	6 (54.5%)	2 (50.0%)
Amox–Clavulanic Acid	23 (35.9%)	–	12 (36.4%)	–	6 (60.0%)	5 (45.5%)	2 (50.0%)
Ampicillin	–	–	4 (12.1%)	–	4 (40.0%)	2 (18.2%)	2 (50.0%)
Ampicillin–Sulbactam	–	–	–	18 (72.0%)	–	–	–
Trimethoprim–Sulfamethoxazole	26 (40.6%)	–	21 (63.6%)	14 (56.0%)	5 (50.0%)	6 (54.5%)	1 (25.0%)
Doxycycline	39 (60.9%)	–	21 (63.6%)	22 (88.0%)	2 (20.0%)	6 (54.5%)	2 (50.0%)
Minocycline	–	–	–	22 (88.0%)	–	–	–

Table 3: Antibiotic Susceptibility Pattern – Intermediate (n/%)

Antibiotic	Klebsiella (n=64)	Pseudomonas (n=39)	E. coli (n=33)	Acinetobacter (n=25)	Enterobacter (n=10)	Proteus (n=11)	Citrobacter (n=4)
Amikacin	3 (4.7%)	7 (17.9%)	8 (24.2%)	5 (20.0%)	1 (10.0%)	1 (9.1%)	0 (0.0%)
Gentamicin	1 (1.6%)	5 (12.8%)	8 (24.2%)	7 (28.0%)	1 (10.0%)	2 (18.2%)	1 (25.0%)
Tobramycin	–	9 (23.1%)	–	6 (24.0%)	–	–	–
Netilmicin	–	2 (5.1%)	–	–	–	–	–
Ciprofloxacin	3 (4.7%)	13 (33.3%)	4 (12.1%)	6 (24.0%)	2 (20.0%)	4 (36.4%)	1 (25.0%)
Ceftriaxone	3 (4.7%)	–	7 (21.2%)	–	3 (30.0%)	2 (18.2%)	3 (75.0%)
Cefotaxime	2 (3.1%)	–	4 (12.1%)	–	–	–	–
Ceftazidime	–	–	4 (12.1%)	6 (24.0%)	2 (20.0%)	2 (18.2%)	3 (75.0%)
Cefepime	2 (3.1%)	–	8 (24.2%)	–	2 (20.0%)	2 (18.2%)	0 (0.0%)
Cefoxitin	2 (3.1%)	–	–	–	–	3 (27.3%)	–
Cefuroxime	6 (9.4%)	–	8 (24.2%)	–	2 (20.0%)	4 (36.4%)	3 (75.0%)
Ceftaroline	–	–	7 (21.2%)	–	2 (20.0%)	–	2 (50.0%)
Aztreonam	–	7 (17.9%)	–	–	–	–	–
Ceftazidime + Avibactam	–	13 (33.3%)	–	–	–	–	–
Imipenem	6 (9.4%)	7 (17.9%)	7 (21.2%)	5 (20.0%)	1 (10.0%)	1 (9.1%)	1 (25.0%)
Meropenem	6 (9.4%)	8 (20.5%)	4 (12.1%)	4 (16.0%)	1 (10.0%)	0 (0.0%)	0 (0.0%)
Ertapenem	3 (4.7%)	–	4 (12.1%)	–	1 (10.0%)	4 (36.4%)	2 (50.0%)
Piperacillin–Tazobactam	5 (7.8%)	8 (20.5%)	15 (45.5%)	4 (16.0%)	2 (20.0%)	1 (9.1%)	0 (0.0%)
Amox–Clavulanic Acid	1 (1.6%)	–	6 (18.2%)	–	2 (20.0%)	3 (27.3%)	0 (0.0%)
Ampicillin	–	–	3 (9.1%)	–	2 (20.0%)	1 (9.1%)	0 (0.0%)
Ampicillin–Sulbactam	–	–	–	5 (20.0%)	–	–	–
Trimethoprim–Sulfamethoxazole	5 (7.8%)	–	7 (21.2%)	7 (28.0%)	2 (20.0%)	2 (18.2%)	2 (50.0%)
Doxycycline	1 (1.6%)	–	8 (24.2%)	1 (4.0%)	3 (30.0%)	3 (27.3%)	2 (50.0%)
Minocycline	–	–	–	1 (4.0%)	–	–	–

Table 4: Antibiotic Susceptibility Pattern – Resistant (n/%)

Antibiotic	Klebsiella (n=64)	Pseudomonas (n=39)	E. coli (n=33)	Acinetobacter (n=25)	Enterobacter (n=10)	Proteus (n=11)	Citrobacter (n=4)
Amikacin	43 (67.2%)	9 (23.1%)	7 (21.2%)	11 (44.0%)	1 (10.0%)	2 (18.2%)	2 (50.0%)
Gentamicin	42 (65.6%)	6 (15.4%)	6 (18.2%)	8 (32.0%)	1 (10.0%)	2 (18.2%)	1 (25.0%)
Tobramycin	–	4 (10.3%)	–	2 (8.0%)	–	–	–
Netilmicin	–	0 (0.0%)	–	–	–	–	–
Ciprofloxacin	50 (78.1%)	7 (17.9%)	17 (51.5%)	13 (52.0%)	4 (40.0%)	3 (27.3%)	2 (50.0%)
Ceftriaxone	48 (75.0%)	–	11 (33.3%)	5 (20.0%)	5 (50.0%)	4 (36.4%)	0 (0.0%)
Cefotaxime	40 (62.5%)	–	5 (15.2%)	–	–	–	–
Ceftazidime	–	3 (7.7%)	10 (30.3%)	5 (20.0%)	5 (50.0%)	4 (36.4%)	0 (0.0%)
Cefepime	36 (56.3%)	–	12 (36.4%)	11 (44.0%)	3 (30.0%)	4 (36.4%)	2 (50.0%)
Cefoxitin	41 (64.1%)	–	–	–	–	3 (27.3%)	–
Cefuroxime	38 (59.4%)	–	14 (42.4%)	–	5 (50.0%)	4 (36.4%)	0 (0.0%)
Ceftaroline	–	–	8 (24.2%)	–	2 (20.0%)	–	0 (0.0%)
Aztreonam	–	2 (5.1%)	–	–	–	–	–
Ceftazidime + Avibactam	–	3 (7.7%)	–	–	–	–	–
Imipenem	25 (39.1%)	3 (7.7%)	3 (9.1%)	6 (24.0%)	0 (0.0%)	2 (18.2%)	0 (0.0%)
Meropenem	27 (42.2%)	2 (5.1%)	2 (6.1%)	6 (24.0%)	0 (0.0%)	2 (18.2%)	0 (0.0%)
Ertapenem	34 (53.1%)	–	4 (12.1%)	–	0 (0.0%)	2 (18.2%)	0 (0.0%)
Piperacillin–Tazobactam	40 (62.5%)	5 (12.8%)	11 (33.3%)	16 (64.0%)	1 (10.0%)	4 (36.4%)	2 (50.0%)
Amox–Clavulanic Acid	40 (62.5%)	–	15 (45.5%)	–	2 (20.0%)	3 (27.3%)	2 (50.0%)
Ampicillin	–	–	26 (78.8%)	–	4 (40.0%)	8 (72.7%)	2 (50.0%)
Ampicillin–Sulbactam	–	–	–	2 (8.0%)	–	–	–
Trimethoprim–Sulfamethoxazole	33 (51.6%)	–	5 (15.2%)	4 (16.0%)	3 (30.0%)	3 (27.3%)	1 (25.0%)
Doxycycline	24 (37.5%)	–	4 (12.1%)	2 (8.0%)	5 (50.0%)	2 (18.2%)	0 (0.0%)
Minocycline	–	–	–	2 (8.0%)	–	–	–

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