**Original Research Article**

**Genotypic Analysis of *Klebsiella Pneumoniae* among patients admitted in critical care settings in a tertiary care center of Bangladesh**

**Abstract**

Objective: *Klebsiella pneumoniae*, a multidrug-resistant pathogen, is a leading cause of respiratory infections in critically ill patients, particularly those in intensive care units (ICUs). The increasing prevalence of carbapenemase-producing strains, including KPC, NDM, and OXA-48, has significantly limited treatment options and contributed to high mortality rates. This study aims to investigate the antibiotic resistance patterns of K. *pneumoniae* and identify the responsible resistance genes.

Methods: A single-center cross-sectional study was conducted at Chittagong Ma-O-Shihsu Medical College, Bangladesh, from January to March 2024. Endotracheal aspirates were collected from ICU patients undergoing mechanical ventilation. Biochemical assays and phenotypic tests were used for bacterial identification, and antimicrobial susceptibility was assessed using the modified Kirby-Bauer disc diffusion method. Conventional polymerase chain reaction (PCR) was employed to detect resistance genes (KPC, OXA-48, NDM, QnrB, AacB, and Sul-2). Data were analyzed using SPSS version 25.

Results: A high prevalence of antibiotic resistance was observed, particularly against ampicillin, cefuroxime, and cefotaxime. Ceftazidime-avibactam exhibited a lower resistance rate, while colistin resistance was minimal. Carbapenemase gene production was detected in 86% (KPC), 96% (OXA-48), and 74% (NDM) of isolates. Additional resistance genes, including qnrb (80%) and sul-2 (88%), were also prevalent. Mortality among infected patients was approximately 50%.

Conclusion: The study highlights the severe antibiotic resistance pattern and high mortality rate associated with K. *pneumoniae* infections in ICU patients in Bangladesh. The findings underscore the urgent need for stringent antibiotic stewardship and enhanced surveillance to curb the further spread of resistance.

Keywords: *Klebsiella pneumoniae*, antibiotic resistance, resistance genes, ICU,

**Introduction**

Worldwide, infections continue to be one of the leading causes of mortality associated with intensive care units(1). In critically ill patients, respiratory infections, particularly ventilation-associated pneumonia (VAP) and community-acquired pneumonia (CAP), are prevalent and can be life-threatening. which is largely associated with *Klebsiella pneumoniae*, a multidrug-resistant organism. The species typically infects humans by integrating into the human gastrointestinal microbiota, although it also colonizes the respiratory tract. The high rate of acute infection is a direct result of the organism's extensive virulence spectrum, which is explicitly attributed to the plasmid-associated gene (2). Additionally, the bacteria's polysaccharide vesicles enable it to significantly evade the immune system. Furthermore, the organism swiftly acquires the extended-spectrum beta lactamase or carbapenemase gene, which results in resistance to third-generation cephalosporin or carbapenem. This significantly reduces the treatment options (3). In addition, there is a growing number of strains that are reported to produce carbapenemases of functional class A (KPC), class B (NDM), and class D (OXA-48), as well as co-producing more than one type of carbapenems (4). Wyres et al.(5) have demonstrated that antibiotic resistance is linked to specific genetic determinants for distinct genetic lineages of the organism. By comprehending the resistance pattern, it is possible to prevent the development of additional resistance.
There has been a twofold increase in the reporting of ESKAPE (*Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter spp.*) pathogens between 2015 and 2018 in Bangladesh, indicating an increase in the prevalence of multi-drug-resistant K. pneumonia (6). Their dissemination has been further facilitated by the absence of awareness, monitoring, and antibiotic stewardship. Additionally, the prevalence of pandrug-resistant carbapenemase-resistant K pneumonia in Bangladesh has increased by nearly 14% (7). Their increasing gene resistance is further exacerbated by factors such as horizontal gene transfers and transposition of genes. Delhi Metallo-beta-lactamase (NDM), oxacillinase (OXA), and sulfhydryl variables are the most prevalent (6). It is imperative to comprehend the resistant pattern of this organism due to the ever-increasing hazard of resistance, the vulnerability of critical care patients, and the limited treatment options. The objective of this investigation was to examine the resistant pattern of K. pneumonia in an intensive care unit and identify the gene that is responsible for the resistance.

**Materials & Methods**

This single-center cross-sectional study was conducted at Chittagong Ma-O-Shihsu Medical College in Chattogram, Bangladesh, from January 2024 to March 2024. Endotracheal aspirates were obtained from ICU patients who were undergoing mechanical ventilation. The organism was identified through biochemical assays, colony morphology, and other phenotypic characteristics following inoculation in Triple Sugar Iron (Himedia, India), Motility Indole Urea Iron (Himedia, India),, and Citrate agar media Iron (Himedia, India), The final selection consisted of fifty samples. Antibiotic discs were categorized according to the WHO AWaRE (Access, Watch, Reserve) classification(8). The WHO Aware Category, concentration and abbreviation can be found in supplementary table 1.

All isolates were tested for antimicrobial susceptibility using modified Kirby-Bauer disc diffusion on Mueller–Hinton agar plates (Himedia, India) according to Clinical and Laboratory Standards Institute (CLSI 2023) guidelines. Antibiotic discs (6 mm diameter) were obtained from BD BBL™ Sensi-Disc™ (Becton Dickinson, Franklin Lakes, NJ, USA) and placed on the inoculated plates at a distance of 24 mm from each other to prevent overlapping of inhibition zones. Following 24-hour incubation at 37°C, the diameter of the inhibition zones was measured and interpreted according to CLSI breakpoints.

Isolates showing resistance to meropenem or imipenem underwent confirmatory testing using the modified Carbapenem Inactivation Method (mCIM) and EDTA-Carbapenem Inactivation Method (eCIM) to differentiate between serine carbapenemases and metallo-β-lactamases. Briefly, a loopful of bacterial culture was suspended in 2 mL tryptic soy broth (TSB) (Himedia, India) , and a 10 μg meropenem disk was immersed in this suspension for 4 hours at 35°C. The disk was then placed on a Mueller-Hinton agar plate (Himedia, India) inoculated with E. coli ATCC 25922, and incubated for 18-24 hours. Carbapenemase production was confirmed by a zone diameter of ≤15 mm.

 To exclude other potential respiratory flora, specimens showing mixed growth were subcultured on selective media (MacConkey agar with crystal violet; Himedia, India) to isolate pure *K. pneumoniae* colonies. The final selection consisted of fifty confirmed *K. pneumoniae* isolates.The virulence primers for thermal cycler amplification were identified using conventional polymerase chain reaction (PCR) for the following genes: Kpc(9), Oxa 48(9), Ndm(10), Qnrb(11), Aadb(12), and Sul-2(13). For DNA extraction we followed the boiling method. 2-3 colonies were briefly suspended in 100 μL of sterile distilled water and then boiled for 10 minutes. Afterwards they were centrifuged and the supernatant served as the DNA template. We performed PCR following gene-specific primers with appropriate cycling conditions. First electrophoresis was done on a 1.5% agarose gel through 1X TAE buffer and ethidium bromide staining. Afterwards, the PCR products were identified using ultraviolet transillumination. SPSS version 25 was employed to aggregate and analyze the data.

**Results**

Table 1 presents the antibiotic susceptibility results of the 50 *K. pneumoniae* isolates, organized according to the WHO AWaRE classification. The highest resistance rates were observed against Access antibiotics, particularly ampicillin (100%) and amoxicillin-clavulanate (88%). Among Watch antibiotics, cefuroxime (96%), cefotaxime (94%), and ceftazidime (92%) demonstrated high resistance rates, while meropenem and imipenem showed resistance rates of 76% and 74%, respectively. For Reserve antibiotics, ceftazidime-avibactam had a lower resistance rate (42%), while colistin showed the lowest resistance (2%), with only one isolate showing complete resistance.

The resistance pattern of *K. pneumonia* is demonstrated in figure 1. Ampicillin showed complete resistance in all 50 isolates. Most antibiotics, including amoxiclav, cotrimoxazole, cephalosporins (cefuroxime, ceftriaxone, cefixime), carbapenems (imipenem, meropenem), and fluoroquinolones (levofloxacin, ciprofloxacin), had resistance rates over 80% (≥40 isolates). However, colistin showed only 1 resistant isolate and 47 susceptible isolates (94%). Ceftazidime-avibactam combination shows relatively better effectiveness compared to most other antibiotics tested, with 22 sensitive isolates (44%). Tigecycline demonstrated moderate activity with 36 resistant isolates, 5 intermediate, and 9 sensitive isolates (72%,10%, 18% respectively)

The distribution of resistance genes among *K. pneumoniae* isolates is shown in Figure 3. aadB gene was isolated in all the samples (100%). Afterwareds, OXA-48 was the most prevalent gene (48, 96%), followed by KPC (43, 86%) and NDM (37, 74%). Other resistance genes were also commonly detected: sul-2 (44, 88%), qnrB (40, 80%). Multiple resistance genes were present in most isolates.

**Discussion**

This is one of the first studies to investigate the antibiotic-resistant pattern and potential resistant gene of *K. pneumonia* in Bangladesh, where the information is still limited.
Our study demonstrates that critical care patients are particularly susceptible to *K. pneumonia* infection. In our investigation, infection esulted in the deaths of nearly half of the patients. Our discovery is comparable to a meta-analysis that identified a 47.66% mortality rate for carbapenem-resistant K. pneumonia (14). The treatment option is severely restricted and the mortality rate is further exacerbated by the propensity for the rapid development of resistance to the most commonly used antibiotics(15).
A study from China also highlighted increased mortality in ICU settings, particularly among elderly patients with carbapenem-resistant infections, suggesting that patient characteristics and hospital environments both contribute to adverse outcomes(16).

All samples in our study exhibit a high rate of resistance to ampicillin, cefuroxime, and cefotaxime, as well as multidrug resistance. The combination of ceftazidime and avibactam exhibited a lower resistant rate, while colistin only exhibited one instance of complete resistance. Our results are comparable to those of Aminul et al. (17) who identified a resistant pattern that is nearly identical to ours. Similar results are also reflected from a recent study in Ethiopia that reported complete resistance to several first-line antibiotics, including cefotaxime and cefazolin, with similarly high resistance to ceftriaxone (84%)(18). Our resistance rates also exceed those reported in a Saudi Arabian long-term care facility, which found resistance to cotrimoxazole (75.9%), ciprofloxacin (65.5%), and gentamicin (62.1%), suggesting potentially more severe resistance patterns in our ICU setting.(19)

On the other hand, the combination of ceftazidime-avibactam demonstrated lower resistance (56%, 28/50) compared to most other antibiotics tested in our study, highlighting its potential value as a therapeutic option for these extensively resistant infections. This finding is consistent with recent literature that has found ceftazidime-avibactam as a viable treatment for infections caused by carbapenemase-producing *K. pneumoniae*, particularly those expressing KPC enzymes(20). However, the 56% resistance rate observed in our study is concerning, as ceftazidime-avibactam is considered a reserve antibiotic.

Colistin had the minimal resistance in our study. This low resistance rate is consistent with findings from other studies and can be attributed to the restricted use of colistin in ICU settings due to concerns about nephrotoxicity(21). However, recent reports from India have documented increasing colistin resistance (up to 60%) in hospital-acquired *K. pneumoniae* isolates(22), highlighting a potentially worrying trend that may eventually impact Bangladesh.

The production of carbapenemase genes was observed in 86% (KPC), 96% (Oxa 48), and 74% (NDM) of the samples. Aminul et al. [10] discovered NDM in 23.34% of samples, OXA-48 in 8%, and KPC in 11% of samples. The significantly increased gene production in comparison to other studies may be attributed to the specific selection of ICU admitted patients. NDM is a gene that is relatively noble and has been endemic to India, Pakistan, and Bangladesh, which has allowed it to develop a unique resistant pattern (Lee et al., 2016). This gene confers resistance to virtually all β-lactams and is easily transferable via plasmids, explaining its rapid dissemination.

The co-occurrence of multiple carbapenemase genes in our isolates is especially concerning. The simultaneous presence of KPC, OXA-48, and NDM severely limits treatment options and reflects findings from an Neonatal Intensive Care Unit (NICU) outbreak study that documented co-existence of blaKPC-2 and blaNDM-1 genes in *K. pneumoniae* isolates(23).

The high prevalence of non-β-lactam resistance genes, including aadB (100%), qnrB (80%), and sul-2 (88%), further explains the multidrug-resistant nature of our isolates. Ballén et al. (24) similarly attributed extensive drug resistance and aminoglycoside resistance to the aadB gene. The qnrB gene, responsible for fluoroquinolone resistance, was detected in 80% of our isolates, consistent with the high ciprofloxacin resistance (92%) observed phenotypically. Sul-2 was present in 88% of our samples, substantially higher than the 43% reported by Kashefieh et al.(25) in co-trimoxazole-resistant Klebsiella isolates, suggesting regional variations in resistance determinants.

Demographic factors may also contribute to the observed resistance patterns. Studies from China and other regions have identified advanced age, particularly above 60 years) as a significant risk factor for carbapenem-resistant *K. pneumoniae* infections(16). While our study did not specifically analyze age-related patterns, the ICU setting itself represents a high-risk environment for resistance development and transmission, as documented in multiple studies worldwide(26).

**Conclusion**

Our findings have significant implications for antimicrobial stewardship and infection control in Bangladesh. The presence of multiple resistance mechanisms in our ICU isolates reflects a critical need for enhanced surveillance, stricter infection control measures, and responsible antibiotic use. The identification of specific resistance genes provides valuable information for developing targeted interventions and diagnostic approaches. The study emphasizes the high mortality rate and significant antibiotic resistance pattern in critically ill patients in Bangladesh. It is imperative to exercise antibiotic stewardship in order to prevent the emergence of additional resistance.

**Recommendations**

Our study findings highlight the urgent need for comprehensive strategies to combat antimicrobial resistance in *Klebsiella pneumoniae*. The ONE HEALTH approach offers an integrated framework particularly suited to addressing this challenge. This approach recognizes that the health of humans, animals, and the environment are intrinsically interconnected and interdependent(27). For AMR specifically, this perspective acknowledges that resistant organisms can spread rapidly through healthcare facilities, animals, food systems, and environmental reservoirs and requires comprehensive and evidence based tailored interventions to combat AMR.

**Limitations**

This investigation was undertaken on a highly specific population and was conducted at a single center with only descriptive analysis. Therefore, the outcome may not be applicable in other contexts and may not be generalizable. In order to generate more generalizable findings, it is necessary to conduct future research at multiple centers with a diverse array of patients. Additionally, longitudinal studies are needed to track resistance evolution and transmission dynamics over time.

Consent

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

Disclaimer (Artificial intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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Table 1: Antibiotics and gene production frequency of the Klebsiella

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Category | Antibiotic | Susceptibility | KPC | OXA-48 | NDM | qnrB | aadB | Sul-2 |
|  |  |  | Absent | Present | Absent | Present | Absent | Present | Absent | Present | Present | Absent | Present |
| ACCESS | Ampicillin | Resistant | 7(14%) | 43(86%) | 2(4%) | 48(96%) | 13(26%) | 37(74%) | 10(20%) | 40(80%) | 50(100%) | 6(12%) | 44(88%) |
| Amoxiclav | Resistant | 7(14%) | 37(74%) | 2(4%) | 42(84%) | 9(18%) | 35(70%) | 10(20%) | 34(68%) | 44(88%) | 4(8%) | 40(80%) |
| Sensitive | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 0 | 1(2%) | 1(2%) | 1(2%) | 0 |
| MS | 0 | 5(10%) | 0 | 5(10%) | 3(6%) | 2(4%) | 0 | 5(10%) | 5(10%) | 1(2%) | 4(8%) |
| Cotrimazole | Resistant | 6(12%) | 1(2%) | 2(4%) | 42(84%) | 0 | 34(68%) | 10(20%) | 34(68%) | 44(88%) | 2(4%) | 41(82%) |
| Sensitive | 2(4%) | 38(76%) | 1(2%) | 4(8%) | 9(18%) | 2(4%) | 0 | 5(10%) | 5(10%) | 2(4%) | 3(6%) |
| MS | 0 | 3(5%) | 0 | 1(2%) | 4(8%) | 1(2%) | 0 | 1(2%) | 1(2%) | 2(4%) | 0 |
| WATCH | Ciprofloxacin | Resistant | 6(12%) | 40(80%) | 2(4%) | 44(88%) | 10(20%) | 36(72%) | 10(20%) | 36(72%) | 46(92%) | 6(12%) | 40(80%) |
| Sensitive | 1(2%) | 2(4%) | 0 | 3(6%) | 2(4%) | 1(2%) | 0 | 3(6%) | 3(6%) | 0 | 3(6%) |
| MS | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| Gentamycin | Resistant | 6(12%) | 37(74%) | 2(4%) | 41(82%) | 9(18%) | 34(68%) | 10(20%) | 33(66%) | 43(86%) | 5(10%) | 38(76%) |
| Sensitive | 1(2%) | 5(10%) | 0 | 6(12%) | 3(6%) | 3(6%) | 0 | 6(12%) | 6(12%) | 1(2%) | 5(10%) |
| MS | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| Ceftazidime | Resistant | 6(12%) | 31(62%) | 1(2%) | 36(72%) | 9(18%) | 28(56%) | 9(18%) | 28(56%) | 37(74%) | 6(12%) | 31(62%) |
| Sensitive | 1(2%) | 12(24%) | 1(2%) | 12(24%) | 1(2%) | 12(24%) | 1(2%) | 12(24%) | 13(26%) | 0 | 13(26%) |
| Cefotaxime | Resistant | 7(14%) | 43(86%) | 2(4%) | 48(96%) | 13(26%) | 37(74%) | 10(20%) | 40(80%) | 50(100%) | 6(12%) | 44(88%) |
| Piperacillin+Tazobactam | Resistant | 7(14%) | 37(74%) | 2(4%) | 42(84%) | 10(20%) | 34(68%) | 10(20%) | 34(68%) | 44(88%) | 6(12%) | 38(76%) |
| Sensitive | 0 | 5(10%) | 0 | 5(10%) | 2(4%) | 3(6%) | 0 | 5(10%) | 5(10%) | 0 | 5(10%) |
| MS | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| Ceftriaxone | Resistant | 6(12%) | 42(84%) | 2(4%) | 46(92%) | 12(24%) | 36(72%) | 10(20%) | 38(76%) | 48(96%) | 6(12%) | 42(84%) |
| Sensitive | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| MS | 1(2%) | 0 | 0 | 1(2%) | 1(2%) | 0 | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| Cefixime | Resistant | 7(14%) | 42(84%) | 2(4%) | 47(94%) | 13(26%) | 36(72%) | 10(20%) | 39(78%) | 49(98%) | 6(12%) | 43(86%) |
| Sensitive | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| Cefepime | Resistant | 7(14%) | 42(84%) | 2(4%) | 47(94%) | 13(26%) | 39(78%) | 10(20%) | 39(78%) | 49(98%) | 6(12%) | 43(86%) |
| Sensitive | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| Cefuroxime | Resistant | 7(14%) | 43(86%) | 2(4%) | 48(96%) | 13(26%) | 37(74%) | 10(20%) | 40(80%) | 50(100%) | 6(12%) | 44(88%) |
| Levofloxacin | Resistant | 6(12%) | 39(78%) | 2(4%) | 43(86%) | 10(20%) | 35(70%) | 10(20%) | 35(70%) | 45(90%) | 5(10%) | 40(80%) |
| Sensitive | 1(2%) | 3(6%) | 0 | 4(8%) | 3(6%) | 1(2%) | 0 | 4(8%) | 4(8%) | 0 | 4(8%) |
| MS | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 1(2%) | 0 |
| RESERVE | Colistin | Resistant | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 1(2%) |
| Sensitive | 7(14%) | 40(80%) | 2(4%) | 45(90%) | 12(24%) | 35(70%) | 10(20%) | 37(74%) | 47(94%) | 6(12%) | 47(94%) |
| MS | 0 | 2(4%) | 0 | 2(4%) | 1(2%) | 1(2%) | 0 | 2(4%) | 2(4%) | 0 | 2(4%) |
| Cefta+avibac | Resistant | 2(4%) | 26(52%) | 2(4%) | 26(52%) | 6(12%) | 22(44%) | 7(14%) | 21(42%) | 28(56%) | 4(8%) | 24(48%) |
| Sensitive | 0 | 22(44%) | 0 | 22(44%) | 7(14%) | 15(30%) | 3(6%) | 19(38%) | 22(44%) | 2(4%) | 20(40%) |
| Imipenam | Resistant | 6(12%) | 35(70%) | 2(4%) | 39(78%) | 6(12%) | 35(70%) | 8(16%) | 33(66%) | 41(82%) | 5(10%) | 36(72%) |
| Sensitive | 1(2%) | 7(14%) | 0 | 8(16%) | 6(12%) | 2(4%) | 1(2%) | 7(14%) | 8(16%) | 1(2%) | 7(14%) |
| MS | 0 | 1(2%) | 0 | 1(2%) | 1(2%) | 0 | 1(2%) | 0 | 1(2%) | 0 | 1(2%) |
| Meropenam | Resistant | 6(12%) | 37(74%) | 2(4%) | 41(82%) | 8(16%) | 35(70%) | 9(18%) | 34(68%) | 43(86%) | 5(10%) | 38(76%) |
| Sensitive | 1(2%) | 6(12%) | 0 | 7(14%) | 5(10%) | 2(4%) | 1(2%) | 6(12%) | 7(14%) | 1(2%) | 6(12%) |
| Amikacin | Resistant | 6(12%) | 39(78%) | 2(4%) | 43(86%) | 9(18%) | 36(72%) | 10(20%) | 35(70%) | 45(90%) | 5(10%) | 40(80%) |
| Sensitive | 1(2%) | 4(8%) | 0 | 5(10%) | 4(8%) | 1(2%) | 0 | 5(10%) | 5(10%) | 1(2%) | 4(8%) |
| Tigecycline | Resistant | 6(12%) | 30(60%) | 2(4%) | 34(68%) | 9(18%) | 27(54%) | 6(12%) | 30(60%) | 36(72%) | 3(6%) | 33(66%) |
| Sensitive | 0 | 4(8%) | 0 | 4(8%) | 1(2%) | 3(6%) | 2(4%) | 2(4%) | 4(8%) | 1(2%) | 3(6%) |
| MS | 1(2%) | 10(20%) | 0 | 9(18%) | 3(6%) | 7(14%) | 2(4%) | 8(16%) | 10(20%) | 2(4%) | 8(16%) |

Figure 1: Resistance pattern of *Klebsiella* to different antibiotics

Figure 2: Frequency of gene production of *Klebsiella* samples

Figure 3: Outcome of the infected patients

Supplementary table 1: Distribution of WHO AWaRe antibiotic classification

|  |  |  |  |
| --- | --- | --- | --- |
| **Category** | **Antibiotic** | **Abbreviation** | **Concentration** |
| **Access** | Ampicillin | AMP | 10 μg |
|  | Amoxicillin-clavulanate | AMC | 20/10 μg |
|  | Cefazolin | CZ | 30 μg |
| **Watch** | Cefuroxime | CXM | 30 μg |
|  | Cefotaxime | CTX | 30 μg |
|  | Ceftazidime | CAZ | 30 μg |
|  | Ciprofloxacin | CIP | 5 μg |
|  | Gentamicin | GM | 10 μg |
|  | Piperacillin-tazobactam | TZP | 100/10 μg |
|  | Meropenem | MEM | 10 μg |
|  | Imipenem | IPM | 10 μg |
| **Reserve** | Ceftazidime-avibactam | CZA | 30/20 μg |
|  | Colistin | CT | 10 μg |
|  | Polymyxin B | PB | 300 units |