

ORIGINAL RESEARCH

DISTRIBUTION OF KLEBSIELLA PNEUMONIAE ISOLATED FROM CLINICAL SPECIMENS OF INTENSIVE CARE UNIT PATIENTS AND THEIR ANTIMICROBIAL SUSCEPTIBILITY PROFILE: A RETROSPECTIVE STUDY FROM A TERTIARY CARE HOSPITAL IN GUJARAT

Minal Joshi ^{1*}, Komal Dholaria ¹, Malhar Madariya ², Vishal Pandya ³, Jaydev Pandya ⁴

¹ Assistant Professor, Department of Microbiology, GMERS Medical College and Hospital, Junagadh, Gujarat, India

² Assistant Professor, Department of Emergency Medicine, GMERS Medical College and Hospital, Junagadh, Gujarat, India

³ Associate Professor, Department of Microbiology, GMERS Medical College and Hospital, Junagadh, Gujarat, India

⁴ Professor and Head, Department of Microbiology, GMERS Medical College and Hospital, Junagadh, Gujarat, India

*Correspondence: Minal Joshi <dr.minaltrivedi@gmail.com>

ABSTRACT

Background: *Klebsiella pneumoniae* is a leading cause of healthcare-associated infection in intensive care units (ICUs), and the spread of extended-spectrum β -lactamase- and carbapenemase-producing strains has steadily narrowed the therapeutic options. Local surveillance of its distribution and susceptibility pattern is essential for empirical prescribing and antibiotic policy. **Objective:** To determine the distribution of *K. pneumoniae* isolated from clinical specimens of ICU patients and to describe its antimicrobial susceptibility profile. **Methods:** A retrospective, record-based cross-sectional study was conducted in the Department of Microbiology of a tertiary care teaching hospital in Gujarat. All clinical specimens received from ICU patients between July 2025 and July 2026 were reviewed. Specimens had been processed by standard methods, including Gram staining and culture on blood, chocolate and MacConkey agar, with identification by conventional biochemical tests. Antimicrobial susceptibility had been determined by the modified Kirby–Bauer disc diffusion method on Mueller–Hinton agar and interpreted according to Clinical and Laboratory Standards Institute (CLSI) criteria. Demographic, specimen and susceptibility data were retrieved from laboratory records and the WHONET database and summarised descriptively. **Results:** Of 2,730 ICU specimens, 475 (17.4%) yielded bacterial growth, and *K. pneumoniae* accounted for 98 (20.6%) of the culture-positive specimens. Fifty-six isolates (57.1%) came from men and 42 (42.9%) from women; the 56–75-year age group contributed the most isolates (39; 39.8%), followed by the 36–55-year group (23; 23.5%). Lower respiratory tract specimens accounted for 41 isolates (41.8%), wound swabs for 27 (27.6%), urine for 11 (11.2%) and blood for 7 (7.1%). Susceptibility was highest to tobramycin (94%), amikacin (90%), meropenem (89%), netilmicin (84%) and gentamicin (82%), and lowest to amoxicillin-clavulanate (16%), cefazolin (21%), cefuroxime (32%), ciprofloxacin (42%) and cefoperazone and ceftazidime (48% each). **Conclusion:** *K. pneumoniae* was responsible for one in five culture-positive ICU specimens, chiefly from older men and from the respiratory tract. Susceptibility to aminoglycosides and meropenem was well preserved, whereas most cephalosporins, amoxicillin-clavulanate and fluoroquinolones performed poorly. Continued surveillance, susceptibility-guided prescribing and strict infection-control practice are needed to contain resistant *K. pneumoniae* in the ICU.

Keywords: *Klebsiella pneumoniae*; Intensive care unit; Antimicrobial susceptibility; Antibiotic resistance; Kirby–Bauer; WHONET

Introduction

Klebsiella pneumoniae is a Gram-negative, encapsulated, non-motile, lactose-fermenting, facultatively anaerobic bacillus of the family Enterobacteriaceae (now Enterobacterales). First isolated by Carl Friedländer in 1882 from the lungs of patients who had died of pneumonia, it is widely distributed in soil, surface water and the gastrointestinal tract of humans and animals, and it readily colonises the hospital environment [1,2]. Clinically it is among the commonest causes of both

community- and hospital-acquired infection, classically producing pneumonia, septicaemia and urinary tract infection but also a wide range of other pyogenic infections [3].

The organism is intrinsically resistant to ampicillin [4], and over the past three decades the proportion of isolates producing extended-spectrum β -lactamases (ESBLs) has risen steadily, frequently accompanied by co-resistance to fluoroquinolones and aminoglycosides. These resistance determinants are carried on plasmids, transposons and integron-borne gene cassettes, which move freely between strains and species [5,6]. Carbapenems have long been the agents of choice for infections caused by multidrug-resistant (MDR) *K. pneumoniae*, but the emergence and global dissemination of carbapenemase-producing, carbapenem-resistant *K. pneumoniae* (CRKP) has eroded even this option [7]. The organism also persists in surface waters and on environmental surfaces, which favours its spread within healthcare settings [8,9]. Among ICU patients the picture is compounded by the convergence of high-risk resistant clones with hypervirulent lineages, leaving clinicians with fewer effective drugs and patients with higher morbidity and mortality [10].

Intensive care units are the epicentre of this problem. Invasive devices, prolonged ventilation, complex tubing, heavy exposure to broad-spectrum antibiotics and the serious comorbidities of critically ill patients together make ICU patients far more likely than ward patients to acquire infection with MDR Gram-negative organisms [11]. Because the prevalence and susceptibility profile of *K. pneumoniae* vary considerably between hospitals, regions and time periods, locally generated surveillance data are indispensable for rational empirical therapy and for the formulation of institutional antibiotic policy.

The present study was therefore undertaken to determine the distribution of *K. pneumoniae* isolated from various clinical specimens of ICU patients at a tertiary care teaching hospital in Gujarat and to describe the antimicrobial susceptibility profile of these isolates.

Materials and Methods

Study design and setting

This retrospective, record-based cross-sectional study was conducted in the Department of Microbiology of GMERS Medical College and Hospital, Junagadh, a tertiary care teaching hospital in Gujarat, India. Laboratory records of all clinical specimens received from patients admitted to the ICUs of the hospital between July 2025 and July 2026 were reviewed.

Ethical considerations

The study involved analysis of existing clinical and laboratory data generated during routine patient care; no patient contact, intervention or additional sampling took place. The protocol was approved by the Institutional Ethics Committee, which waived the requirement for individual informed consent in view of the retrospective, record-based design. **[Ref number: IEC n/89/2026]** All records were de-identified before analysis, and confidentiality was maintained throughout. The study was conducted in accordance with the Declaration of Helsinki and applicable national ethical guidelines.

Inclusion and exclusion criteria

All non-duplicate *Klebsiella pneumoniae* isolates obtained from clinical specimens of patients admitted to the adult, paediatric, and neonatal intensive care units during the study period were included. Only the first isolate of *K. pneumoniae* from each patient was considered; repeat isolates of the same organism from the same patient were excluded. Isolates of organisms other than *K. pneumoniae* and isolates obtained from outpatients or non-ICU inpatients were excluded.

Specimen processing and identification

Specimens were processed according to the laboratory's standard operating procedures. Each specimen was examined by Gram stain and inoculated onto blood agar, chocolate agar and MacConkey agar, which were incubated at 37 °C for 24 hours. *K. pneumoniae* was suspected from the characteristic large, dome-shaped, mucoid, lactose-fermenting colonies on

MacConkey agar and confirmed by a standard battery of biochemical tests, including indole, methyl red, Voges–Proskauer, citrate utilisation, urease, triple sugar iron and motility, following established procedures [2].

Antimicrobial susceptibility testing

Antimicrobial susceptibility was determined on Mueller–Hinton agar by the modified Kirby–Bauer disc diffusion method [12], and zone diameters were interpreted according to the current CLSI M100 breakpoints [13]. Commercial discs (HiMedia Laboratories Pvt. Ltd., Mumbai, India) of the following agents were used: amoxicillin-clavulanate (20/10 µg), piperacillin-tazobactam (100/10 µg), cefazolin (30 µg), cefuroxime (30 µg), cefoperazone (75 µg), ceftazidime (30 µg), ceftriaxone (30 µg), cefepime (30 µg), aztreonam (30 µg), meropenem (10 µg), gentamicin (10 µg), amikacin (30 µg), tobramycin (10 µg), netilmicin (30 µg), ciprofloxacin (5 µg), levofloxacin (5 µg), trimethoprim-sulfamethoxazole (1.25/23.75 µg), tetracycline (30 µg) and nitrofurantoin (300 µg). Nitrofurantoin was tested and reported only for urinary isolates. Not every agent was tested against every isolate; the number of isolates tested therefore varies by antibiotic, and susceptibility percentages are calculated with the number tested as the denominator.

Data collection

Demographic details (age and sex), ICU location, specimen type and antimicrobial susceptibility results for each isolate were retrieved from laboratory registers and the WHONET antimicrobial resistance surveillance software [14], into which the laboratory enters all culture and susceptibility results prospectively.

Sample size

The minimum number of ICU clinical specimens to be reviewed was estimated using the single-proportion formula, $n = Z^2pq/d^2$, where $Z = 1.96$ for a 95% confidence level, p was the expected proportion, $q = 1 - p$, and d was the absolute precision (0.05). The expected proportion of *K. pneumoniae* among ICU clinical specimens (67.9%; 93/137) was obtained from Alshuqayfi et al. Substitution of these values yielded a minimum sample size of 335 specimens. After allowing 10% for incomplete records, the required sample size was 370 specimens. All 2,730 ICU clinical specimens received during the study period were reviewed, exceeding the calculated minimum sample size. The 98 *K. pneumoniae* isolates identified constituted the subgroup subsequently analysed.

Statistical analysis

Data were compiled in Microsoft Excel 2019 and analysed in SPSS version 19.0 (SPSS Inc., Chicago, IL, USA). The analysis was descriptive: categorical variables (sex, age group, specimen type and susceptibility category) are presented as counts and percentages. Percentages for demographic and specimen distributions use the 98 isolates as the denominator; susceptibility percentages use the number of isolates tested against each agent.

Results

Isolation rate

During the study period 2,730 clinical specimens from ICU patients were processed for culture and susceptibility testing. Bacterial growth was obtained from 475 (17.4%), and *K. pneumoniae* was isolated from 98 of these, a proportion of 20.6% of culture-positive specimens and 3.6% of all ICU specimens received.

Demographic distribution

Of the 98 isolates, 56 (57.1%) were from male and 42 (42.9%) from female patients. The largest number came from patients aged 56–75 years (39; 39.8%), followed by those aged 36–55 years (23; 23.5%). Neonates under one month of age accounted for 13 isolates (13.3%), and patients over 75 years for 7 (7.1%) (Table 1).

Table 1. Distribution of *K. pneumoniae* isolates by sex and age (N = 98)

Characteristic	Category	n	%
Sex	Male	56	57.1
	Female	42	42.9
Age	< 1 month	13	13.3
	1 month – 15 years	7	7.1
	16–35 years	9	9.2
	36–55 years	23	23.5
	56–75 years	39	39.8
	76–95 years	7	7.1
	Total	98	100.0

Percentages recalculated from the counts; the submitted table gave sex as 57%/42% and age as 13/7/9/23/39/7%. An empty 96–105-year row has been removed.

Specimen distribution

Lower respiratory tract specimens—tracheal aspirates (n=22), sputum (n=17), and endotracheal secretions (n=2)—together accounted for 41 isolates (41.8%), followed by wound swabs (27; 27.6%). (Table 2).

Table 2. Distribution of *K. pneumoniae* isolates by specimen type (N = 98)

Specimen	n	%
Tracheal aspirate	22	22.4
Sputum	17	17.3
Endotracheal secretion	2	2.0
Wound swab	27	27.6
Urine	11	11.2
Blood	7	7.1
Drain fluid	7	7.1
Pus	3	3.1
Other body fluid	2	2.0
Total	98	100.0

Percentages recalculated from the counts. The percentage column of the submitted Table 2 (37.2, 19, 18.2, 16.1, 5.8, 2.2, 0.7, 0.7) did not correspond to its own frequency column and appears to have been carried over from another source; the counts, which sum to 98, have been retained.

Antimicrobial susceptibility

The susceptibility of the 98 isolates to the agents tested is shown in Table 3 and Figure 1. Susceptibility was highest to the aminoglycosides—tobramycin (94%), amikacin (90%), netilmicin (84%) and gentamicin (82%)—and to meropenem (89%). Piperacillin-tazobactam retained activity against three-quarters of isolates (75%), and cefepime against two-thirds (68%). Susceptibility to the third-generation cephalosporins was modest (ceftriaxone 59%, ceftazidime and cefoperazone 48% each), as it was to aztreonam (57%), trimethoprim-sulfamethoxazole (67%) and tetracycline (58%). The fluoroquinolones performed poorly (levofloxacin 51%, ciprofloxacin 42%), and susceptibility was lowest to cefuroxime (32%), cefazolin (21%) and amoxicillin-clavulanate (16%). Among urinary isolates, 54% were susceptible to nitrofurantoin.

Table 3. Antimicrobial susceptibility of *K. pneumoniae* isolates from ICU patients (N = 98)

Antimicrobial agent	Susceptible (%)
Amoxicillin-clavulanate	16
Piperacillin-tazobactam	75
Cefazolin	21
Cefuroxime	32
Cefoperazone	48
Ceftazidime	48
Ceftriaxone	59
Cefepime	68
Aztreonam	57
Meropenem	89
Gentamicin	82
Amikacin	90
Tobramycin	94
Netilmicin	84
Ciprofloxacin	42
Levofloxacin	51
Trimethoprim-sulfamethoxazole	67
Tetracycline	58
Nitrofurantoin (urinary isolates only)	54

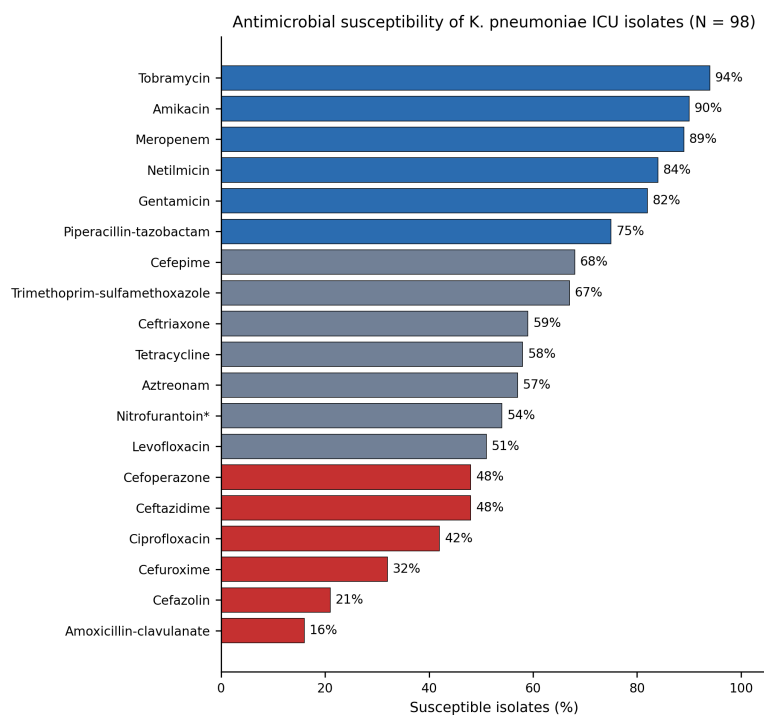


Figure 1. Antimicrobial susceptibility of *K. pneumoniae* isolates from ICU patients (N = 98). Bars show the percentage of isolates susceptible to each agent; *nitrofurantoin was tested on urinary isolates only.

Discussion

In this one-year review of ICU specimens from a tertiary care hospital in Gujarat, *K. pneumoniae* accounted for one in five culture-positive specimens. The isolates came predominantly from older men and from the lower respiratory tract, and while susceptibility to aminoglycosides and meropenem was largely preserved, the older β -lactams, amoxicillin-clavulanate and the fluoroquinolones had lost much of their activity.

The proportion of culture-positive ICU specimens yielding *K. pneumoniae* (20.6%) is close to the 19.2% reported by Santella et al. from a university hospital in southern Italy [16]. Considerably higher figures have been reported from some centres: Paray and Kaur found *K. pneumoniae* in 66.5% of ICU isolates in a north Indian tertiary hospital [17], and Alshuqayfi et al. recorded a proportion of 67.9% in a Saudi ICU [15]. Such variation reflects differences in the case mix, the range of specimens submitted, the presence of other dominant ICU pathogens such as *Acinetobacter baumannii* and *Pseudomonas aeruginosa*, and local infection-control practice, and underlines why institution-specific data are needed.

The male predominance observed here (57.1%) closely matches that reported by Akter et al. from Bangladesh (57% male, 42% female) [18] and is a consistent finding in ICU series, probably reflecting the sex distribution of ICU admissions rather than any biological susceptibility. The concentration of isolates in the 56–75-year age group is likewise expected: older patients have more comorbidity, longer ICU stays and greater exposure to invasive devices and antibiotics, and Santella et al. similarly found the highest isolation rates in patients over 60 years [16]. The appreciable contribution of neonates (13.3%) in the present series is noteworthy, since *K. pneumoniae* is a leading cause of neonatal sepsis in Indian neonatal ICUs and is frequently ESBL-producing in that setting; the susceptibility pattern of neonatal isolates may merit separate analysis in future work.

Lower respiratory tract specimens were the commonest source of *K. pneumoniae* (41.8%), followed by wound swabs (27.6%) and urine (11.2%). This distribution is characteristic of the ICU, where mechanical ventilation, endotracheal intubation and impaired airway clearance predispose to ventilator-associated pneumonia, and it agrees with the predominance of respiratory isolates described in other ICU series [15,16]. Sarathbabu et al., working across all hospital departments, also found sputum to be a major source of *K. pneumoniae*, alongside urine and pus [19]. The relatively high share of wound swabs in the present series may reflect the surgical and trauma case load of the ICUs studied.

The susceptibility pattern has direct implications for empirical prescribing. Amoxicillin-clavulanate (16%), cefazolin (21%) and cefuroxime (32%) can no longer be regarded as reliable options for ICU *K. pneumoniae* infections, and susceptibility to the third-generation cephalosporins of around 50% is consistent with ESBL production in roughly half of the isolates, although ESBL status was not confirmed phenotypically in this study. Fluoroquinolone susceptibility of 42–51% mirrors the well-documented co-selection of quinolone resistance with ESBL carriage [5,6]. Similar losses of cephalosporin and fluoroquinolone activity have been reported from other Indian centres [17,19,20], and Alshuqayfi et al. described an even more resistant profile in Saudi ICU isolates [15]. By contrast, aminoglycoside susceptibility of 82–94% in the present series is higher than in many contemporary reports and suggests that amikacin and tobramycin remain useful companion agents locally. Piperacillin-tazobactam (75%) and cefepime (68%) retained moderate activity.

Meropenem susceptibility of 89% is a reassuring finding, since national surveillance through the Indian Council of Medical Research antimicrobial resistance network has documented a steady fall in carbapenem susceptibility among *K. pneumoniae* isolates from Indian tertiary hospitals over the past decade, with ICU isolates the most affected [21]. The 11% of isolates non-susceptible to meropenem nevertheless represent probable carbapenemase producers and warrant confirmatory testing, because such strains are associated with high mortality and very limited treatment options [7]. Gastrointestinal colonisation is a recognised precursor of subsequent infection with resistant *K. pneumoniae* in hospitalised patients [22], and the capacity of the organism to acquire and transmit resistance genes to other species means that control of CRKP in the ICU depends as much on hand hygiene, contact precautions and screening as on antibiotic selection.

Limitations

This was a single-centre retrospective study based on laboratory records, and clinical details such as the indication for ICU admission, duration of stay, device use, prior antibiotic exposure and outcome were not available, so no distinction could be made between infection and colonisation and no risk-factor analysis was possible. Susceptibility was determined by disc diffusion alone; minimum inhibitory concentrations were not measured, and phenotypic or molecular confirmation of ESBL and carbapenemase production was not undertaken. Not all agents were tested against all isolates, and results for imipenem, ertapenem and colistin were not available. The relatively small number of isolates limits the precision of the susceptibility estimates.

Conclusion

K. pneumoniae accounted for about one-fifth of culture-positive ICU specimens at this hospital, predominantly from older male patients and from the respiratory tract. The isolates showed poor susceptibility to amoxicillin-clavulanate, first- and second-generation cephalosporins and fluoroquinolones and only moderate susceptibility to third-generation cephalosporins, while aminoglycosides, meropenem and piperacillin-tazobactam retained good activity. These data can inform the institutional antibiotic policy for empirical treatment of suspected *K. pneumoniae* infection in the ICU. Regular surveillance of susceptibility patterns, de-escalation guided by culture results, and rigorous infection-control practice are needed to limit the emergence and spread of resistant strains.

Source of funding

None.

Conflicts of Interest

None declared.

References

1. Podschun R, Ullmann U. *Klebsiella* spp. as nosocomial pathogens: epidemiology, taxonomy, typing methods, and pathogenicity factors. *Clin Microbiol Rev.* 1998;11(4):589-603.
2. Winn WC Jr, Allen SD, Janda WM, Koneman EW, Procop GW, Schreckenberger PC, et al. *Koneman's color atlas and textbook of diagnostic microbiology*. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2006.
3. Sastry AS, Bhat S, editors. *Ananthanarayan and Paniker's textbook of microbiology*. 9th ed. Hyderabad: Universities Press; 2013.
4. Murray PR, Holmes B, Aucken HM. Enterobacteriaceae. In: Borriello SP, Murray PR, Funke G, editors. *Topley and Wilson's microbiology and microbial infections: bacteriology*. 10th ed. Vol. 2. London: Hodder Arnold; 2005.
5. Kang HY, Jeong YS, Oh JY, Tae SH, Choi CH, Moon DC, et al. Characterization of antimicrobial resistance and class 1 integrons found in *Escherichia coli* isolates from humans and animals in Korea. *J Antimicrob Chemother.* 2005;55(5):639-44.
6. Stalder T, Barraud O, Casellas M, Dagot C, Ploy MC. Integron involvement in environmental spread of antibiotic resistance. *Front Microbiol.* 2012;3:119.
7. Nordmann P, Cuzon G, Naas T. The real threat of *Klebsiella pneumoniae* carbapenemase-producing bacteria. *Lancet Infect Dis.* 2009;9(4):228-36.
8. Podschun R, Pietsch S, Höller C, Ullmann U. Incidence of *Klebsiella* species in surface waters and their expression of virulence factors. *Appl Environ Microbiol.* 2001;67(7):3325-7.
9. Chang D, Sharma L, Dela Cruz CS, Zhang D. Clinical epidemiology, risk factors, and control strategies of *Klebsiella pneumoniae* infection. *Front Microbiol.* 2021;12:750662.
10. Piperaki ET, Syrogiannopoulos GA, Tzouveleakis LS, Daikos GL. *Klebsiella pneumoniae*: virulence, biofilm and antimicrobial resistance. *Pediatr Infect Dis J.* 2017;36(10):1002-5.
11. Peleg AY, Hooper DC. Hospital-acquired infections due to gram-negative bacteria. *N Engl J Med.* 2010;362(19):1804-13.
12. Bauer AW, Kirby WM, Sherris JC, Turck M. Antibiotic susceptibility testing by a standardized single disk method. *Am J Clin Pathol.* 1966;45(4):493-6.

13. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 35th ed. CLSI supplement M100. Wayne, PA: CLSI; 2025.
14. Stelling JM, O'Brien TF. Surveillance of antimicrobial resistance: the WHONET program. *Clin Infect Dis*. 1997;24 Suppl 1:S157-68.
15. Alshuqayfi KA, Elhaj YHA, Albanghali MA, Alharbi RA, Sindi AAA, Aljadani S, et al. *Klebsiella pneumoniae* isolates from the intensive care unit at South Qunfudah Hospital in Saudi Arabia: an emerging antimicrobial resistance profile. *Infect Drug Resist*. 2025;18:2451-60.
16. Santella B, Boccella M, Folliero V, Iervolino D, Pagliano P, Fortino L, et al. Antimicrobial susceptibility profiles of *Klebsiella pneumoniae* strains collected from clinical samples in a hospital in Southern Italy. *Can J Infect Dis Med Microbiol*. 2024;2024:5548434.
17. Paray AA, Kaur A. Antimicrobial susceptibility pattern and phenotypic detection of metallo-beta lactamases in *K. pneumoniae* isolates in a tertiary care hospital. *Int J Res Med Sci*. 2022;10(10):2173-8.
18. Akter J, Chowdhury AMMA, Al Forkan M. Study on prevalence and antibiotic resistance pattern of *Klebsiella* isolated from clinical samples in South East region of Bangladesh. *Am J Drug Discov Dev*. 2014;4(1):73-9.
19. Sarathbabu R, Ramani TV, Rao KB, Panda S. Antibiotic susceptibility pattern of *Klebsiella pneumoniae* isolated from sputum, urine and pus samples. *IOSR J Pharm Biol Sci*. 2012;1(2):4-9.
20. Namitha BN, Kumar SS, Pasha RSA. The antibiogram of *Klebsiella pneumoniae* isolated from clinical samples in various clinical settings at a South Indian tertiary care hospital. *Indian J Microbiol Res*. 2026;13(1):126-32.
21. Indian Council of Medical Research. Annual report: Antimicrobial Resistance Research and Surveillance Network, January 2023 to December 2023. New Delhi: ICMR; 2024.
22. Qin X, Hu F. Gastrointestinal carriage of *Klebsiella pneumoniae* is a risk factor of subsequent hospital acquired infection. *J Emerg Crit Care Med*. 2017;1:47.