e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(7); 145-152

Original Research Article

Distribution and Antibiotic Resistance Pattern of Gram-Negative Nonfermenters in Critical Care Unit of a Tertiary Care Teaching Hospital in Eastern Part of India

Biswas Sriparna[1], Pathak Mrinmoy[2], Patra Sanjit Kumar[3], Sarkar Aditya Prasad[4]

[1]Senior Resident, Department of Microbiology, Bankura Sammilani Medical College and Hospital, India
[2]Assistant Professor, Department of Microbiology, Bankura Sammilani Medical College and Hospital, India
[3]Professor & Head, Department of Microbiology, Bankura Sammilani Medical College and Hospital, India
[4]Professor & Head, Department of Community Medicine, Bankura Sammilani Medical College and Hospital, India

Received: 01-04-2026 / Revised: 15-05-2026 / Accepted: 21-06-2026
Corresponding author: Dr. Sriparna Biswas
Conflict of interest: Nil

Abstract

Background: Non-fermenting gram-negative bacilli (NFGNB) are important nosocomial pathogens associated with significant morbidity due to their multidrug resistance (MDR) and production of metallo-β-lactamases (MBL). This study aimed to evaluate the distribution, antimicrobial resistance patterns, and MBL production among NFGNB isolates in a tertiary care hospital.

Methods: This cross-sectional study included 1750 clinical samples collected from patients aged 12 years onward admitted in Critical Care Unit (CCU) and were processed in the microbiology laboratory, of which 155 NFGNB isolates were identified. Organisms were identified using standard microbiological techniques. Antimicrobial susceptibility testing was performed using the VITEK 2 compact system ((bioMérieux) as per CLSI guidelines. MBL production by the isolates were also detected by Vitek 2 compact system.

Results: Out of 1750 samples, 155 (8.86%) were identified as NFGNB**.** Pseudomonas aeruginosa (n=52) and Acinetobacter baumannii (n=44) were the predominant organisms, mainly isolated from sputum samples. Carbapenem resistance was notably high in Acinetobacter baumannii compared to Pseudomonas aeruginosa. MBL production was detected in 72.73% of Acinetobacter baumannii and 36.54% of Pseudomonas aeruginosa, with all MBL producers being multidrug-resistant. Colistin and minocycline showed relatively better susceptibility across most isolates.

Conclusion: A high prevalence of MDR and MBL-producing NFGNB, particularly Acinetobacter baumannii, was observed. The increasing resistance to commonly used antibiotics and reliance on last-resort drugs highlight the urgent need for antimicrobial stewardship, strict infection control, and continuous surveillance.

Keywords: NFGNB, MBL, MDRO, Pseudomonas aeruginosa, Acinetobacter baumannii.

DOI: 10.25258/ijcpr.18.7.24

Introduction

Non-fermenting Gram-negative bacilli (NFGNB) comprise a taxonomically heterogeneous group of organisms that either do not utilize carbohydrates for energy or metabolize them solely through oxidative pathways. [1] NFGNBs are widely distributed in the environment and can persist throughout healthcare settings, colonize on different medical equipment, parenteral fluids, distilled water, and sinks, largely due to their inherent resistance to commonly used disinfectants. [2] Immunocompromised patients, including those with HIV/AIDS, diabetes, malignancy patients receiving chemotherapy, transplant recipients, patients on corticosteroids, CCU patients, are at increased risk of NFGNB-associated healthcare infections. [3] This diverse group comprises organisms such as Pseudomonas spp., Acinetobacter spp., Stenotrophomonas maltophilia, and the Burkholderia cepacia complex (BCC) etc. At present, Pseudomonas aeruginosa and Acinetobacter baumannii are the most frequently isolated human pathogens among these, while infections due to others are seen less frequently. [4] In recent years, the indiscriminate use of

antimicrobials has led to the emergence of NFGNB as significant healthcare-associated pathogens. They are implicated in a wide range of infections, including bacteremia, meningitis, pneumonia, urinary tract infection, surgical site infection, osteomyelitis, wound infections etc. [5] NFGNB account for approximately 12–16% of bacterial isolates obtained from various clinical specimens. [1] Multidrug-resistant organisms (MDROs), including NFGNB, exhibit resistance to one or more classes of antimicrobial agents, such as β-lactams (including penicillins, cephalosporins, monobactams, carbapenems), fluoroquinolones, and aminoglycosides. [6] NFGNB demonstrate a high prevalence of multidrug resistance, mediated by the production of diverse β-lactamases such as extended-spectrum β-lactamases (ESBL), AmpC β-lactamases, and metallo-β-lactamases (MBL, resulting in the emergence of strains resistant to nearly all commonly used antimicrobial agents. [7]

Prior exposure to antimicrobial therapy is one of the most significant risk factors for the development of such resistant pathogens, underscoring the importance of continuous surveillance of bacterial etiologies and infection patterns to guide appropriate treatment, limit resistance development, and reduce hospital-acquired cross-infections. [8] All these reasons highlights the urgent need for continuous surveillance and the development of novel therapeutic strategies. [9] This study aimed to isolate and identify NFGNB from clinical samples of patients admitted in Critical Care Unit (CCU) and to characterize their antimicrobial resistance patterns. These findings helped to understand the epidemiology of NFGNB-associated infections. Moreover, ongoing surveillance of resistance trends also helped clinicians in optimizing antimicrobial therapy, promoting cost-effective patient care and to prevent the emergence/re-emergence and spread of multidrug resistant NFGNB in healthcare settings.

Materials and Methods

Study setting and data collection: This is an Observational Descriptive study with Cross sectional design, which was conducted for a time period of two and a half year (January 1, 2023-June 31, 2025) in the Department of Microbiology at a tertiary care teaching hospital of Eastern India. Patients aged 12 years onward admitted in Critical Care Unit (CCU) were evaluated for this study. After getting the necessary clearance from the Institute Ethics Committee (BSMC/IEC/3362/2022) the process of data collection was started. Data were collected via interview and laboratory test results. Collected data were codified and entered in MS Excel Spreadsheet.

Sample collection and processing: Blood (5-10ml) was collected aseptically in automated blood culture bottle (BacT/ALERT/ 3D/60 for adults). Other clinical samples (e.g., urine, pus, wound swab, sputum, pleural fluid etc.) were collected aseptically in sterile container. All the samples were collected with proper labelling. Samples were disposed to the department of Microbiology without any delay. The samples were received carefully after checking the details. These samples were collected before administration of antibiotics.

Blood samples in automated blood culture bottles were incubated in BACT/ALERT/ 3D/60 and after giving positive signal from the machine, blood sample from automated blood culture bottle was subculture in sheep blood agar and MacConkey agar media (Hi-Media) and incubated aerobically at 37°C for overnight. Blood culture bottle was incubated maximum up to seven days, if any positive signal is not given within seven days the bottles were discarded.

Other samples were directly inoculated in sheep blood agar and MacConkey agar and incubated aerobically at 37°C for overnight. After overnight incubation they were processed according to standard microbiological protocols for identification and antibiogram pattern testing.

Bacterial identification and AST: The identification and antimicrobial susceptibility testing were done using the VITEK 2 compact system (bioMérieux) as per the Clinical and Laboratory Standards Institute (CLSI) recommendation. Identification was done by using VITEK 2 GN ID cards. Isolates were simultaneously processed by reagent card ASN406 on Vitek 2 compact system (bioMérieux) for their antimicrobial susceptibility testing. MBL production by the isolates were also detected by Vitek 2 compact system.

Statistical analysis: Statistical analysis was performed using Microsoft Excel and IBM SPSS Statistics Version 22. Data comparison and study results were finalized using these tools. A p-value of less than 0.05 was considered statistically significant.

Results

Between January 1, 2023 and June 31, 2025, a total of 1750 samples were received from critical care unit for culture and sensitivity.

The most common samples received from CCU was urine sample (625, 35.71%) followed by blood (450, 25.71%), sputum (375, 21.43%), pus (135,7.71%), body fluids (70, 4%), ET aspirate (65, 3.71%) and foley's catheter tip (30,1.71%) (Table1). A higher proportion of urine samples suggests that urinary tract infections (UTIs) are one

of the most commonly encountered infections in CCU patients. This is followed by blood samples, which indicates the clinical significance of bacteremia and septicemia in patients admitted to the CCU. Body fluids including pleural, cerebrospinal, and ascitic fluids are collected in cases where invasive infections are suspected.

Out of 1,750 clinical samples, NFGNB were isolated from 155 samples, accounting a prevalence of 8.86%. Among these cases, 90 (58.06%) were from male patients and 65 (41.94%) were from female patients. The patients' median age (IQR) was 57 (24-79).

Out of the 155 NFGNB, Pseudomonas aeruginosa was the most commonly isolated organism (52, 33.55%), followed by Acinetobacter baumannii (44, 28.39%) and Burkholderia cepacia complex (16, 10.32%).

Other isolated NFGNB included Sphingomonas paucimobilis (13, 8.39%), Acinetobacter lwoffii (10, 6.45%), Pseudomonas fluorescens (9, 5.81%), Stenotrophomonas maltophilia (5, 3.22%), Pseudomonas putida (4, 2.58%), and Acinetobacter junii (2, 1.29%) (Table 2)

Isolation rate of NFGNB was highest from sputum sample (46, 29.67%) followed by blood (34, 21.94%), pus (24, 15.48%), urine (21, 13.55%), body fluids (11, 7.1%), ET aspirate (11, 7.1%) and foley's catheter tip (8, 5.16%). This indicates that NFGNB were responsible for most of the respiratory tract infections and also played an important role causing blood stream infection in CCU patients (Table 3).

Pseudomonas aeruginosa was mainly isolated from sputum (n=15), urine (n=12), and pus samples (n=8), while Acinetobacter baumannii showed a higher distribution in sputum (n=14), blood (n=7), and ET aspirates (5). Burkholderia cepacia complex was predominantly isolated from sputum (9) and blood (4). Sphingomonas paucimobilis was most commonly found in blood (5) and pus samples (4). Other NFGNB, such as Acinetobacter lwoffii were most commonly found in ET aspirates (4), while Pseudomonas fluorescens and Stenotrophomonas maltophilia were more frequently isolated from blood samples. Lesser isolates included Pseudomonas putida and Acinetobacter junii, which were found in small numbers across sputum, blood, and urine samples (Table 4).

The NFGNB demonstrated a high burden of multidrug resistance across isolated organisms. Pseudomonas aeruginosa exhibited a moderate to high resistance pattern, with 57.7% resistance to β-lactam/β-lactamase inhibitor combinations (ticarcillin-clavulanic acid and piperacillin-tazobactam) and ceftazidime, while resistance to cefepime was slightly lower (48.1%) and cefoperazone-sulbactam showed comparatively better sensitivity (50%). Carbapenem resistance was observed in 36.5% of isolates (imipenem, meropenem and doripenem). Aminoglycosides showed relatively lower resistance, with amikacin at 34.6% and gentamicin at 32.7%. Fluoroquinolone resistance ranged from 40.4% (levofloxacin) to 44.2% (ciprofloxacin). Among the tetracyclines, minocycline demonstrated lower resistance (26.9%), whereas tigecycline showed complete resistance due to intrinsic resistance property. Colistin remained the most effective agent with minimal resistance (5.8%), indicating its retained efficacy against Pseudomonas aeruginosa. Acinetobacter baumannii also showed a markedly high level of multidrug resistance, with resistance to β-lactam/β-lactamase inhibitor combinations ranging from 54.5% (cefoperazone-sulbactam) to 79.5% (piperacillin-tazobactam), and similarly high resistance to cephalosporins such as ceftazidime (77.3%) and cefepime (65.9%). Carbapenem resistance was also higher at 72.7% for imipenem, meropenem, and doripenem, indicating widespread carbapenem resistance. Aminoglycoside resistance was also significant, with amikacin at 72.7% and gentamicin at 65.9%. Fluoroquinolones showed high resistance rates, with ciprofloxacin at 72.7% and levofloxacin relatively lower at 59.1%. Among tetracyclines, minocycline demonstrated comparatively low resistance (20.5%), and tigecycline showed moderate resistance (43.2%). Colistin remained the most effective agent with minimal resistance (2.3%), highlighting its role as a last-resort drug for Acinetobacter baumannii infections. Burkholderia cepacia complex exhibited high resistance to most of the antibiotics, including cotrimoxazole (75%). Other NFGNB isolates also showed very high resistance. Pseudomonas fluorescens and Pseudomonas putida demonstrated resistance rates around 75–100% across most antibiotics. Acinetobacter lwoffii and Acinetobacter junii also showed high resistance, frequently up to 100%, with relatively better susceptibility to minocycline and colistin. Sphingomonas paucimobilis showed moderate to high resistance, with better activity of minocycline (15.4%) and colistin (30.8%). Stenotrophomonas maltophilia demonstrated intrinsic carbapenem resistance but retained lower resistance to cotrimoxazole, fluoroquinolones and minocycline (20% each). Although the sample sizes for Pseudomonas putida (n=4), Stenotrophomonas maltophilia (n=5) and Acinetobacter junii (n=2) were very small which limits the generalization of their resistance pattern findings (Table 5). The prevalence of Metallo-beta-lactamases (MBL) production and multidrug resistance was highest in Acinetobacter baumannii (72.73%). Notably, all MBL-producing isolates of Pseudomonas

aeruginosa (36.54%) and Acinetobacter baumannii (72.73%) were multidrug-resistant, whereas in B. cepacia complex, a slightly lower proportion of isolates were MDR (50%) compared to MBL producers (56.25%) (Table 6).

Table 1: Distribution of Clinical Samples

Type of sample	Number of Samples (n=1750)	Percentage (%)
Urine	625	35.72
Blood	450	25.72
Sputum	375	21.43
Pus	135	7.71
Body fluids	70	4
ET Aspirate	65	3.71
Foley's Catheter tip	30	1.71
Total	**1750**	**100**

Table 2: Prevalence of NFGNB Isolates

Name of isolate	Numbers of isolates	Percentage (%)
Pseudomonas aeruginosa	52	33.55
Acinetobacter baumannii	44	28.39
Burkholderia cepacia complex	16	10.32
Sphingomonas paucimobilis	13	8.39
Acinetobacter lowffii complex	10	6.45
Pseudomonas fluroscence	9	5.81
Strenotrophomonas maltophilia	5	3.22
Pseudomonas putida	4	2.58
Acinetobacter junii	2	1.29
Total	**155**	**100**

Table 3: Prevalence of NFGNB in Different Clinical Samples

Sample Type	Number of Isolates (n)	Percentage (%)
Sputum	46	29.67
Blood	34	21.94
Pus	24	15.48
Urine	21	13.55
Body Fluids	11	7.10
ET Aspirate	11	7.10
Foley’s Catheter Tip	8	5.16
Total	**155**	**100**

Table 4: Distribution of NFGNB in Different Clinical Specimens

Name of Isolate	Sputum	Blood	Pus	Urine	Body fluids	ET Aspirate	Foley's Catheter tip	Total
Pseudomonas aeruginosa	15	7	8	12	6	2	2	**52**
Acinetobacter baumannii	14	7	6	4	3	5	5	**44**
Burkholderia cepacia complex	9	4	3	0	0	0	0	**16**
Sphingomonas paucimobilis	1	5	4	2	1	0	0	**13**
Acinetobacter lowffii complex	2	2	0	0	1	4	1	**10**
Pseudomonas fluroscence	3	3	1	2	0	0	0	**9**
Strenotrophomonas maltophilia	0	3	2	0	0	0	0	**5**
Pseudomonas putida	2	2	0	0	0	0	0	**4**
Acinetobacter junii	0	1	0	1	0	0	0	**2**
Total	**46**	**34**	**24**	**21**	**11**	**11**	**8**	**155**

Table 5: Resistance Pattern of Isolated NFGNB

Name of Antibiotic	Pseudomonas aeruginosa	Pseudomonas fluorescens	Pseudomonas putida	Acinetobacter baumannii	Acinetobacter lwoffii	Acinetobacter junii	Burkholderia cepacia complex	Sphingomonas paucimobilis	Strenotrophomonas maltophilia
Ticaracillin/ Clavulanic Acid	30 (57.7%)	7 (77.8%)	4 (100%)	34 (77.3%)	10 (100%)	2 (100%)	11 (68.8%)	9 (62.9%)	3 (60%)
Piperacillin/ Tazobactum	30 (57.7%)	7 (77.8%)	4 (100%)	35 (79.5%)	10 (100%)	2 (100%)	11 (68.8%)	8 (61.5%)	3 (60%)
Ceftazidime	30 (57.7%)	7 (77.8%)	4 (100%)	34 (77.3%)	10 (100%)	2 (100%)	9 (56.3%)	8 (61.5%)	3 (60%)
Cefoperazone/ Sulbactam	26 (50%)	6 (66.7%)	2 (50%)	24 (54.5%)	8 (80%)	1 (50%)	3 (18.8%)	5 (38.5%)	3 (60%)
Cefepime	25 (48.1%)	5 (55.6%)	3 (75%)	29 (65.9%)	9 (90%)	2 (100%)	10 (62.5%)	5 (38.5%)	3 (60%)
Aztreonam	25 (48.1%)	8 (88.9%)	4 (100%)	31 (70.5%)	10 (100%)	2 (100%)	12 (75%)	11 (84.6%)	3 (60%)
Doripenem	19 (36.5%)	5 (55.6%)	4 (100%)	32 (72.7%)	7 (70%)	2 (100%)	11 (68.8%)	6 (46.2%)	5 (100%)
Imipenem	19 (36.5%)	4 (44.4%)	4 (100%)	32 (72.7%)	7 (70%)	2 (100%)	11 (68.8%)	6 (46.2%)	5 (100%)
Meropenem	19 (36.5%)	4 (44.4%)	3 (75%)	32 (72.7%)	7 (70%)	2 (100%)	7 (43.8%)	7 (53.8%)	5 (100%)
Amikacin	18 (34.6%)	4 (44.4%)	2 (50%)	32 (72.7%)	5 (50%)	2 (100%)	9 (56.3%)	4 (30.8%)	3 (60%)
Gentamicin	17 (32.7%)	4 (44.4%)	4 (100%)	29 (65.9%)	3 (30%)	2 (100%)	11 (68.8%)	6 (46.2%)	3 (60%)
Ciprofloxacin	23 (44.2%)	7 (77.8%)	4 (100%)	32 (72.7%)	7(70%)	2 (100%)	7 (43.8%)	9 (62.9%)	1 (20%)
Levofloxacin	21 (40.4%)	6(66.7%)	3 (75%)	26 (59.1%)	5(50%)	2 (100%)	7 (43.8%)	6 (46.2%)	1 (20%)
Minocycline	14 (26.9%)	2(22.2%)	1 (25%)	9 (20.5%)	2(20%)	0	7 (43.8%)	2 (15.4%)	1 (20%)
Tigecycline	52 (100%)	9(100%)	4 (100%)	19 (43.2%)	4(40%)	2 (100%)	8 (50%)	4 (30.8%)	1 (20%)
Colistin	3 (5.8%)	1(11.1%)	1 (25%)	1 (2.3%)	1(10%)	0	8 (50%)	4 (30.8%)	1 (20%)
Trimethoprim /Sulfamethoxazole	52 (100%)	9(100%)	4 (100%)	31 (70.5%)	6(60%)	0	12 (75%)	7 (53.8%)	1 (20%)

Table 6: Prevalence of Metallo-B-Lactamase (MBL) Production and Multidrug Resistance in Pseudomonas Aeruginosa, Acinetobacter Baumannii and Burkholderia Cepacia Complex

Name of Organism	MBL Producers	MDR isolates
Pseudomonas aeruginosa (n=52)	19 (36.54%)	19 (36.54%)
Acinetobacter baumannii (n=44)	32 (72.73%)	32 (72.73%)
Burkholderia cepacia complex (n=16)	9 (56.25%)	8 (50%)

Discussion

NFGNB are increasingly recognized as important opportunistic pathogens in critically ill patients due to their intrinsic resistance and ability to survive in hospital environments. In our study, the prevalence of NFGNB was found 8.86% which is in accordance with results of a study done by Samanta et al. [10] Various studies have showed various results in this context. Chang & Huang11 reported that 31.62 % nonfermenter were isolated while Rao and Shivananda [12] reported a higher positivity rate 66.88%. In contrast to that, Malini et al [13] and Bruno et al [14] have reported a lower rate of

isolation i.e. 4.5% and 2.18% respectively. Reason behind these variations in prevalence of NFGNB might be because of the hospital infection control practices of different institutes. [15]

In our study, it was found that majority of NFGNB were isolated from sputum samples (29.67%) which is in accordance with a study conducted by Yadav et al.16 Followed by sputum samples, we found that NFGNB were isolated from blood (21.94%), pus (15.48%), urine (13.55%), body fluids (7.1%), ET aspirate (7.1%) and foley's catheter tip (5.16%). In contrast to, a study was conducted by Bhatnagar et al, where they found that majority of NFGNB were isolated from pus (49.20%) followed by sputum (19.84%), urine (12.69%), bronchial aspirate (9.12%), pleural fluid (3.96%) and blood (1.19%). [17]

In this study, most common NFGNB isolated was Pseudomonas aeruginosa (33.55%), followed by Acinetobacter baumannii (28.39%) which is similar to findings of Juyal et al [5] and Bhatnagar et al. [17] Next to that our study revealed an isolation rate of 10.32% for Burkholderia cepacia complex followed by Sphingomonas paucimobilis (8.39%), Acinetobacter lwoffii (6.45%), Pseudomonas fluorescens (95.81%), Stenotrophomonas maltophilia (3.22%), Pseudomonas putida (2.58%), and Acinetobacter junii (1.29%). On the other hand, Yadav et al have found that most common NFGNB isolate was Acinetobacter baumannii (44.0%) followed by Pseudomonas aeruginosa (40.1%), Burkholderia cepacia complex (8.2%), and Acinetobacter calcoaceticus (2.7%). [16] Sah et al have also found that most common NFGNB isolated was Acinetobacter baumannii. [18]

The present study provides a detailed insight into the distribution and antimicrobial resistance profile of NFGNB in a tertiary care setting of Eastern India. NFGNB were predominantly isolated from respiratory samples, with Pseudomonas aeruginosa (15/52; 28.8%) and Acinetobacter baumannii (14/44; 31.8%) being the most commonly isolated organisms from sputum. This finding is consistent with a study conducted by Joseph et al [19] and Golia et al [20] where they report respiratory specimens, particularly from CCU settings, as the most common source of NFGNB isolation due to ventilator-associated infections and prolonged hospitalization.

Urine (12/52; 23.1%) and pus (8/52; 15.4%) were the next most common sources for Pseudomonas aeruginosa, whereas Acinetobacter baumannii also showed notable isolation from blood (7/44; 15.9%) and endotracheal aspirates (5/44; 11.4%), indicating its strong association with invasive infections such as bacteremia and ventilator-associated pneumonia (VAP). Comparable findings have been reported in Indian tertiary care centres, where Acinetobacter baumannii is increasingly implicated in bloodstream infections and VAP. [21]

Burkholderia cepacia complex was predominantly isolated from sputum (9/16; 56.25%) and blood (4/16; 25%), which indicates its role in opportunistic pulmonary and systemic infections. Less frequently isolated organisms such as Sphingomonas paucimobilis were mainly recovered from blood (5 cases) and pus (4 cases), highlighting their emerging clinical significance. Other NFGNB, including Acinetobacter lwoffii (ET aspirates: 4 cases) and Stenotrophomonas maltophilia (blood predominance), though limited in number, indicate the expanding spectrum of opportunistic pathogens in CCU settings.

A key finding of this study is the high burden of antimicrobial resistance among NFGNB. Pseudomonas aeruginosa demonstrated resistance rates of 57.7% to β-lactam/β-lactamase inhibitor combinations and ceftazidime, while resistance to cefepime was slightly lower (48.1%). Carbapenem resistance was observed in 36.5% of isolates. These findings are comparable to study conducted by Taneja et al [22] and Gill et al [23] where they have reported carbapenem resistance rates ranging from 30% to 60% in Pseudomonas aeruginosa. Aminoglycosides showed relatively better efficacy (amikacin: 34.6%, gentamicin: 32.7% resistance), and minocycline demonstrated the lowest resistance (26.9%). Colistin remained highly effective, with only 5.8% resistance, implying its role as a last-resort drug.

In contrast, Acinetobacter baumannii exhibited significantly higher resistance across all antibiotic classes. Resistance to β-lactam/β-lactamase inhibitor combinations ranged from 54.5% to 79.5%, while cephalosporin resistance was also high (ceftazidime: 77.3%, cefepime: 65.9%). Notably, carbapenem resistance was observed in 72.7% of isolates, which is in concordance with study conducted by Dash et al [24] and Manchanda et al [25] where they found carbapenem resistance rates between 70% and 90%. Aminoglycoside resistance was also substantial (amikacin: 72.7%, gentamicin: 65.9%), and fluoroquinolone resistance ranged from 59.1% to 72.7%. However, minocycline (20.5% resistance) and colistin (2.3% resistance) retained relatively good activity, highlighting limited therapeutic options.

Burkholderia cepacia complex showed high resistance, including 75% resistance to cotrimoxazole, which is traditionally considered the drug of choice. This contrasts with earlier reports where cotrimoxazole susceptibility was relatively preserved, suggesting an emerging resistance trend in this organism. [26] Other NFGNB such as Pseudomonas fluorescens and Pseudomonas putida

demonstrated very high resistance rates (75% to100%) across most antibiotics.

Similarly, Acinetobacter lwoffii and Acinetobacter junii exhibited near-complete resistance, although better susceptibility was noted for minocycline and colistin. However, the small sample sizes (Pseudomonas putida, n=4; Stenotrophomona maltophilia, n=5; Acinetobacter junii, n=2) limit definitive conclusions. Stenotrophomonas maltophilia demonstrated intrinsic carbapenem resistance but relatively lower resistance (20%) to cotrimoxazole, fluoroquinolones, and minocycline, which is consistent with its known susceptibility profile. [27]

The prevalence of MBL production was notably high in Acinetobacter baumannii (32/44; 72.73%) compared to Pseudomonas aeruginosa (19/52; 36.54%). Importantly, all MBL-producing isolates in both species were multidrug-resistant, indicating a strong correlation between MBL production and MDR phenotype. Walsh et al [28] also reported MBL production is a major contributor to carbapenem resistance in NFGNB. In Burkholderia cepacia complex, MBL production (56.25%) slightly exceeded MDR prevalence (50%), suggesting the involvement of additional resistance mechanisms.

Conclusion

The present study highlights the significant clinical burden of NFGNB, particularly Pseudomonas aeruginosa and Acinetobacter baumannii, as major contributors to hospital-acquired infections, predominantly involving the respiratory tract and bloodstream. The findings demonstrate a high prevalence of MDR with Acinetobacter baumannii showing the highest resistance rates, including alarming levels of carbapenem resistance.

The substantial proportion of MBL producers, especially among Acinetobacter baumannii and Pseudomonas aeruginosa, and their strong association with MDR phenotypes, underscores the growing challenge of limited therapeutic options.

While colistin and minocycline retained relatively better efficacy, the emerging resistance even to these last-resort agents is concerning.

The presence of less common NFGNB with high resistance patterns further emphasizes the evolving spectrum of opportunistic pathogens in healthcare settings. Overall, these findings highlight the urgent need for continuous antimicrobial resistance surveillance, strict infection control practices, and implementation of robust antimicrobial stewardship programs.

Future strategies should focus on early detection of resistant strains, rational antibiotic usage, and the development of newer therapeutic options to combat the rising threat of multidrug-resistant NFGNB.

References:

1. Grewal U, Bakshi R, Walia G, Shah P. Antibiotic susceptibility profiles of non-fermenting gram-negative Bacilli at a Tertiary Care Hospital in Patiala, India. Niger Postgrad Med J. 2017;24(2):121. doi:10.4103/npmj.npmj_76_17
2. Dash RK, Mohapatra I, Singh N, Pattnaik D, Panda SS, Smriti S, et al. A Five-Year Trend Analysis of Antibacterial Resistance Patterns Among Non-fermenting Gram-Negative Bacilli: A Retrospective Study from the ICU Settings of a Tertiary Care Hospital. Cureus. 2024 Sep 28. doi:10.7759/cureus.70375
3. Guner Ozenen G, Sahbudak Bal Z, Umit Z, Avcu G, Tekin D, Kurugol Z, et al. Nosocomial Non-fermentative gram negative bacteria bloodstream infections in children; Risk factors and clinical outcomes of carbapenem resistance. Journal of Infection and Chemotherapy. 2021 May;27(5):729–35. doi:10.1016/j.jiac.2020.12.024
4. Fass RJ, Barnishan J, Solomon MC, Ayers LW. In vitro activities of quinolones, beta-lactams, tobramycin, and trimethoprim-sulfamethoxazole against nonfermentative gram-negative bacilli. Antimicrob Agents Chemother. 1996 Jun;40(6):1412–8. doi:10.1128/AAC.40.6.1412
5. Juyal D, Prakash R, Shanakarnarayan S, Sharma M, Negi V, Sharma N. Prevalence of non-fermenting gram negative bacilli and their in vitro susceptibility pattern in a tertiary care hospital of Uttarakhand: A study from foothills of Himalayas. Saudi J Health Sci. 2013;2(2):108. doi:10.4103/2278-0521.117915
6. Chopra I, Schofield C, Everett M, O'Neill A, Miller K, Wilcox M, et al. Treatment of health-care-associated infections caused by Gram-negative bacteria: a consensus statement. Lancet Infect Dis. 2008 Feb;8(2):133–9. doi:10.1016/S1473-3099(08)70018-5 PubMed PMID: 18222164.
7. Gales AC, Jones RN, Forward KR, Liñares J, Sader HS, Verhoef J. Emerging importance of multidrug-resistant Acinetobacter species and Stenotrophomonas maltophilia as pathogens in seriously ill patients: geographic patterns, epidemiological features, and trends in the SENTRY Antimicrobial Surveillance Program (1997-1999). Clin Infect Dis. 2001 May 15;32 Suppl 2:S104-113. doi:10.1086/320183 Pub Med PMID: 11320451.
8. Al Johani SM, Akhter J, Balkhy H, El-Saed A, Younan M, Memish Z. Prevalence of antimicrobial resistance among gram-negative isolates in an adult intensive care unit at a

tertiary care center in Saudi Arabia. Ann Saudi Med. 2010;30(5):364–9. doi:10.4103/0256-4947.67073 PubMed PMID: 20697174; PubMed Central PMCID: PMC2941248.
9. Shelja, Nehra N, Thakran BK, Mor S, Rathee A, Sumedha, Bajera R. To Evaluate The Non-Fermenting Gram Negative Bacilli in A Tertiary Care Hospital, With A Specific Emphasis on Clinical and Microbiological Factors. International J Acad Med Pharm. 2024; 6 (4);792-796
10. Samanta P, Gautam V, Thapar R, Ray P. Emerging resistance of non-fermenting gram negative bacilli in a tertiary care centre. Indian J Pathol Microbiol. 2011;54(3):666. doi:10.4 103/0377-4929.85150
11. Chang TC, Huang AYH. Rapid differentiation of fermentative from nonfermentative gram negative bacilli in positive blood cultures by an impendance method. J Clin Microbiol 2000; 38:3589-94.
12. Rao PS, Shivananda PG. Bacteraemia due to non fermenting gram negative bacilli in immunocompromised patients. Ind J Med Microbiol 1993;11:95-99.
13. Malini A, Deepa E, Gokul B, Prasad S. Nonfermenting Gram-negative bacilli infections in a tertiary care hospital in Kolar, Karnataka. J Lab Physicians 2009;1:62-6.
14. Bruno D, Nishino MK, Priore WN, Remus PR, do Carmo AA, Stefanello VB, et al. Prevalence of Gram-negative non-fermenters patients in Porto Alegre-RS.Jornal Brasileiro de Patologia e Medina Laboratorial 2011;47:529-34.
15. Sidhu S, Arora U, Devi P. Prevalence of nonfermentative Gram negative bacilli in seriously ill patients with bacteraemia. JK Sci 2010;12:168-71.
16. Yadav SK, Bhujel R, Mishra SK, Sharma S, Sherchand JB. Emergence of multidrug-resistant non-fermentative gram negative bacterial infection in hospitalized patients in a tertiary care center of Nepal. BMC Research Notes. 2020 Jul 2;13(1).
17. Bhatnagar R, Kumar S, Bansal G, Mehra S. Identifcation and antimicrobial susceptibility pattern of clinical isolates of non-fermentative gram negative bacilli. Int J Pharma Res Health Sci. 2014;2:347–5.
18. Sah M, Mishra M, Ohora H, Kirikae T, Sherchand J, Rijal B, et al. Nosocomial bacterial infection and antimicrobial resistant pattern in a tertiary care hospital in Nepal. J Inst Med. 2014;36:38–48.
19. Joseph NM, Sistla S, Dutta TK, Badhe AS, Rasitha D, Parija SC. Ventilator-associated pneumonia in a tertiary care hospital in India. J Infect Dev Ctries. 2010;4(4):218–25.
20. Golia S, Sangeetha KT, Vasudha CL. Microbial profile of ventilator-associated pneumonia in a tertiary care hospital. J Clin Diagn Res. 2013;7(8):1629–32.
21. Prashanth K, Badrinath S. Nosocomial infections due to Acinetobacter species. Indian J Med Microbiol. 2006;24(4):284–6.
22. Taneja N, Maharwal S, Sharma M. Imipenem resistance in non-fermenters. Indian J Med Res. 2003;117:92–6.
23. Gill JS, Arora S, Khanna SP, Kumar KH. Multidrug-resistant Pseudomonas aeruginosa. J Glob Infect Dis. 2016;8(4):155–9.
24. Dash M, Padhi S, Mohanty I, Panda P, Parida B. Antimicrobial resistance in ICU pathogens. Indian J Crit Care Med. 2013;17(4):214–9.
25. Manchanda V, Sanchaita S, Singh N. Multidrug resistant Acinetobacter. J Glob Infect Dis. 2010;2(3):291–304.
26. Gautam V, Ray P, Vandamme P, Chatterjee SS, Das A, Sharma K, et al. Identification of Burkholderia cepacia complex. Indian J Med Res. 2009;129(4):409–12.
27. Brooke JS. Stenotrophomonas maltophilia: an emerging pathogen. Clin Microbiol Rev. 2012;25(1):2–41.
28. Walsh TR, Toleman MA, Poirel L, Nordmann P. Metallo-β-lactamases. Clin Microbiol Rev. 2005;18(2):306–25.