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(8); 1558-1562

Original Research Article

Microbiological Profile, Multidrug Resistance Patterns, and Clinical Characteristics of Ventilator-Associated Pneumonia

Milankumar Dharsandia[1], Nawal Pradeep[2], Jigisha Bhoya[3], Meghana Chauhan[4]

[1]Associate Professor, Department of Microbiology, Shantabaa Medical College & General Hospital, Amreli, Gujarat, India

[2]Assistant Professor, Department of Microbiology, JIET Medical College and Hospital, Jodhpur, Rajasthan, India

[3]Assistant Professor, Department of Microbiology, Parul Institute Medical sciences and Research, Parul University, Vadodara, Gujarat, India

[4]Associate Professor and Head, Department of Microbiology, Shantabaa Medical College & General Hospital, Amreli, Gujarat, India

Received: 01-05-2026 / Revised: 15-06-2026 / Accepted: 21-07-2026

Corresponding author: Dr. Milankumar Dharsandia

Conflict of interest: Nil

Abstract

Background: Ventilator-associated pneumonia (VAP) is a major healthcare-associated infection among critically ill patients requiring mechanical ventilation. The increasing prevalence of multidrug-resistant (MDR) pathogens complicates clinical management and limits therapeutic options. Identification of causative organisms, antimicrobial resistance patterns, and patient characteristics is essential to guide empiric therapy and strengthen infection-control practices.

Aim: To determine the occurrence of VAP among mechanically ventilated ICU patients, identify causative bacterial pathogens, assess antimicrobial resistance patterns (including ESBL, AmpC, and MBL production), and describe the clinical characteristics of affected patients.

Methods: A prospective observational study was conducted among 150 adult intensive care unit (ICU) patients requiring invasive mechanical ventilation for 48 hours or more. Patients were monitored prospectively for VAP development. Lower respiratory tract specimens from suspected cases were processed using standard microbiological procedures. Antimicrobial susceptibility testing was performed according to Clinical and Laboratory Standards Institute (CLSI) guidelines. Extended-spectrum beta-lactamase (ESBL), AmpC beta-lactamase, and metallo-beta-lactamase (MBL) production were evaluated.

Results: VAP developed in 15 of 150 mechanically ventilated patients (10.0%). Gram-negative bacilli predominated, accounting for 14 of 15 isolates (93.3%). Klebsiella spp. was the most common pathogen (n = 5), followed by Acinetobacter spp. (n = 3), Pseudomonas spp. (n = 3), Escherichia coli (n = 2), Citrobacter spp. (n = 1), and Staphylococcus spp. (n = 1). Among Gram-negative isolates, ESBL and AmpC production were each detected in 10 isolates (71.4%), while 6 (42.9%) co-produced ESBL and AmpC. MBL production was present in 1 of 8 (12.5%) evaluated Pseudomonas and Klebsiella isolates. High resistance rates to first-line agents were observed among Gram-negative isolates. Frequently observed clinical factors among VAP patients included supine positioning (n = 11, 73.3%), enteral feeding (n = 10, 66.7%), excessive sedation (n = 8, 53.3%), age over 60 years (n = 7, 46.7%), underlying pulmonary pathology (n = 6, 40.0%), Glasgow Coma Scale score under 9 (n = 6, 40.0%), and diabetes mellitus (n = 6, 40.0%).

Conclusion: VAP occurred in 10.0% of mechanically ventilated patients and was primarily caused by multidrug-resistant Gram-negative bacilli. Institutional microbiological surveillance and adherence to evidence-based VAP bundles are critical to optimizing patient outcomes and mitigating resistance.

Keywords: Ventilator-associated pneumonia, multidrug resistance, Gram-negative bacilli, intensive care unit, antimicrobial stewardship.

DOI: 10.25258/ijcpr.18.8.233

Introduction

Ventilator-associated pneumonia (VAP) is defined as nosocomial pulmonary parenchymal infection arising 48 hours or more after initiation of invasive mechanical ventilation.[1,5,6] It remains a major driver of attributable morbidity, prolonged mechanical ventilation, extended intensive care unit

(ICU) stays, and heightened healthcare costs.[1,3,4] Management is increasingly challenged by the rapid global expansion of multidrug-resistant (MDR) pathogens.[6] Pathogenesis involves the breakdown of mechanical airway barriers, micro-aspiration of colonized oropharyngeal secretions, and biofilm formation on endotracheal tubes.[1,4,14] Key clinical risk factors reported in the literature include prolonged ventilation, reintubation, altered mental status, supine positioning, enteral feeding, and prior broad-spectrum antimicrobial exposure.[1,2,7] Gram-negative organisms—most notably Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Escherichia coli—dominate the microbiological spectrum of VAP in many Indian ICUs, alongside Gram-positive pathogens such as Staphylococcus aureus.[8–13] Regional variation in pathogen distribution and antimicrobial resistance patterns underscores the importance of institution-specific microbiological surveillance and antibiograms to guide empirical antimicrobial therapy.[6,7,10,13]

This study aimed to determine the occurrence of VAP in mechanically ventilated ICU patients, characterize the bacterial spectrum and resistance profiles (including ESBL, AmpC, and MBL production), and describe the clinical features of patients developing VAP.

Materials and Methods

Study Design and Eligibility: A prospective observational study was conducted in adult ICUs following approval from the Institutional Ethics Committee. Informed consent was obtained in accordance with institutional ethical guidelines. Enrolled patients were adult ICU admissions (18 years or older) requiring invasive mechanical ventilation for 48 hours or more. Exclusion criteria comprised pre-existing pneumonia at ICU admission, mechanical ventilation under 48 hours, age under 18 years, or incomplete clinical records.

Clinical Surveillance and Diagnosis: Patients were evaluated daily for VAP, defined by new or progressive pulmonary infiltrates on chest radiography combined with two or more clinical criteria: fever (over 38.0°C) or hypothermia (under 36.0°C), leukocytosis (over 12,000 / microliter) or leukopenia (under 4,000 / microliter), purulent endotracheal aspirates, and deteriorating oxygenation. Baseline clinical characteristics—including age, primary diagnosis, comorbidities, Glasgow Coma Scale (GCS) score, positioning, sedation level, enteral feeding, and smoking history—were documented.

Microbiological Evaluation and Phenotypic Resistance Screening: Aseptic endotracheal aspirates were transported immediately for Gram staining and semi-quantitative culture. Clinically significant bacterial isolates were identified to the species level. Antimicrobial susceptibility testing (AST) was executed using Kirby-Bauer disk diffusion methods and interpreted per CLSI criteria. Phenotypic detection of beta-lactamases included:

- **Extended-Spectrum Beta-Lactamases (ESBL):** Screened via ceftazidime and cefotaxime disks alone and in combination with clavulanic acid.
- **AmpC Beta-Lactamases:** Screened using cefoxitin susceptibility and confirmed via inhibitor-based methods.
- **Metallo-Beta-Lactamases (MBL):** Screened among Pseudomonas and Klebsiella isolates using imipenem-EDTA combined disk synergism.

Statistical Analysis: Data were processed using standard statistical software. Categorical parameters were summarized as frequency counts and percentages. Continuous metrics were presented as mean ± standard deviation (SD) or median with interquartile range (IQR) as appropriate.

Results

VAP Occurrence and Pathogen Distribution: Among 150 mechanically ventilated patients, 15 developed VAP, yielding an overall occurrence rate of 10.0% (Table 1). Gram-negative bacilli accounted for 14 of the 15 bacterial isolates (93.3%). Klebsiella spp. was the most common isolate (n = 5, 33.3%), followed by Acinetobacter spp. (n = 3, 20.0%), Pseudomonas spp. (n = 3, 20.0%), E. coli (n = 2, 13.3%), Citrobacter spp. (n = 1, 6.7%), and Staphylococcus spp. (n = 1, 6.7%).

Resistance Phenotypes and Antimicrobial Susceptibility: Among the 14 Gram-negative isolates, ESBL and AmpC production were confirmed in 10 isolates each (71.4%), with dual ESBL + AmpC co-production observed in 6 isolates (42.9%) (Table 2). MBL testing performed on 8 Pseudomonas and Klebsiella isolates identified 1 MBL-positive isolate (12.5%) (Table 3). Gram-negative pathogens demonstrated marked resistance to ampicillin, first- to fourth-generation cephalosporins, and fluoroquinolones (Table 4). No Imipenem resistance was detected among the tested isolates. The single Staphylococcus spp. isolate was fully susceptible to azithromycin, clindamycin, erythromycin, linezolid, and vancomycin.

Clinical Characteristics of VAP Patients: All 15 VAP patients were intubated (100.0%). Frequently documented predisposing factors included supine positioning (n = 11, 73.3%), enteral feeding (n = 10, 66.7%), excessive sedation (n = 8, 53.3%), age over 60 years (n = 7, 46.7%), underlying lung pathology (n = 6, 40.0%), GCS under 9 (n = 6, 40.0%), diabetes mellitus (n = 6, 40.0%), and

cigarette smoking (n = 5, 33.3%) (Table 5).

Table 1: Occurrence of ventilator-associated pneumonia in the study population

Study Population	Number (n)	Percentage (%)
Total mechanically ventilated patients	150	100.0
Patients who developed VAP	15	10.0
Patients who did not develop VAP	135	90.0

Table 2: Distribution of beta-lactamase enzymes among VAP-associated Gram-negative isolates (n = 14)

Resistance Phenotype	Number (n)	Percentage (%)
ESBL Producers	10	71.4
AmpC Producers	10	71.4
ESBL + AmpC Co-Producers	6	42.9
Resistance Phenotype	**Number (n)**	**Percentage (%)**
Total Gram-Negative Isolates	14	100.0

Note: Percentage values exceed 100% due to overlapping resistance mechanisms within individual isolates.

Table 3: Metallo-beta-lactamase (MBL) production among evaluated isolates (n = 8)

Phenotype	Number (n)	Percentage (%)
MBL-Positive Isolates	1	12.5
MBL-Negative Isolates	7	87.5
Total Evaluated (Pseudomonas + Klebsiella)	8	100.0

Table 4: Antimicrobial resistance profiles of VAP isolates [n (%)]

Antibiotic	Klebsiella (n=5)	Acinetobacter (n=3)	Pseudomonas (n=3)	Citrobacter (n=1)	E. coli (n=2)	Staphylococcus (n=1)
Amikacin	2 (40.0)	2 (66.7)	2 (66.7)	1 (100.0)	1 (50.0)	1 (100.0)
Ampicillin	5 (100.0)	3 (100.0)	3 (100.0)	1 (100.0)	2 (100.0)	1 (100.0)
Amoxicillin-clavulanate	3 (60.0)	3 (100.0)	3 (100.0)	0 (0.0)	1 (50.0)	0 (0.0)
Cefazolin	5 (100.0)	3 (100.0)	3 (100.0)	1 (100.0)	2 (100.0)	—
Cefepime	5 (100.0)	3 (100.0)	3 (100.0)	0 (0.0)	2 (100.0)	1 (100.0)
Cefoxitin	3 (60.0)	3 (100.0)	3 (100.0)	1 (100.0)	1 (50.0)	0 (0.0)
Cefotaxime	3 (60.0)	3 (100.0)	3 (100.0)	0 (0.0)	1 (50.0)	—
Ceftazidime	3 (60.0)	3 (100.0)	2 (66.7)	0 (0.0)	1 (50.0)	—
Ceftriaxone	3 (60.0)	3 (100.0)	3 (100.0)	0 (0.0)	1 (50.0)	—
Ciprofloxacin	4 (80.0)	2 (66.7)	3 (100.0)	0 (0.0)	1 (50.0)	0 (0.0)
Cotrimoxazole	4 (80.0)	2 (66.7)	3 (100.0)	1 (100.0)	1 (50.0)	—
Gentamicin	3 (60.0)	2 (66.7)	2 (66.7)	1 (100.0)	1 (50.0)	1 (100.0)
Imipenem	0 (0.0)	0 (0.0)	1 (33.3)	0 (0.0)	0 (0.0)	—
Tetracycline	4 (80.0)	3 (100.0)	3 (100.0)	1 (100.0)	2 (100.0)	—
Azithromycin	—	—	—	—	—	0 (0.0)
Clindamycin	—	—	—	—	—	0 (0.0)
Erythromycin	—	—	—	—	—	0 (0.0)
Linezolid	—	—	—	—	—	0 (0.0)
Vancomycin	—	—	—	—	—	0 (0.0)

Note: Dash (—) indicates the antibiotic was not tested or not clinically indicated for the specified organism group.

Table 5: Clinical characteristics documented among patients with VAP (n = 15)

Clinical Factor	Patients (n)	Percentage (%)
Intubation	15	100.0
Supine positioning	11	73.3
Enteral feeding	10	66.7
Clinical Factor	**Patients (n)**	**Percentage (%)**
Excessive sedation	8	53.3
Age over 60 years	7	46.7
Glasgow Coma Scale under 9	6	40.0
Predisposing acute/chronic pulmonary pathology	6	40.0
Diabetes mellitus	6	40.0
Cigarette smoking history	5	33.3

Discussion

This prospective observational study documented a VAP occurrence rate of 10.0% among adult ICU patients mechanically ventilated for 48 hours or more. This finding is broadly comparable with the variable VAP rates reported in Indian tertiary-care ICUs.[8–12] Differences in VAP incidence across studies may be related to variations in diagnostic criteria, patient populations, severity of illness, duration of mechanical ventilation, surveillance methods, and local infection-control practices.[1,2,5,6]

Gram-negative bacilli dominated the etiological spectrum in the present study, accounting\ for 93.3% of isolates, with Klebsiella spp. being the most common pathogen, followed by Acinetobacter spp. and Pseudomonas spp. A similar predominance of Gram-negative organisms has been reported in Indian ICU studies by Joseph et al., Charles et al., Gupta et al., Patro et al., Patil and Patil, and Chaudhury et al.[8–13] These findings emphasize the important role of Enterobacterales and non-fermenting Gram-negative bacilli in VAP in Indian critical-care settings.

The high prevalence of enzymatic resistance mechanisms—specifically ESBL (71.4%) and AmpC (71.4%) production—in the present study highlights the substantial antimicrobial resistance burden among ICU isolates. Dual ESBL and AmpC co-production was detected in 42.9% of Gram-negative isolates. The high prevalence of resistant Gram-negative organisms in VAP has also been documented in Indian studies, highlighting the therapeutic difficulties associated with MDR pathogens.[8,10,11,13] Although MBL production was detected in only one evaluated isolate (12.5%), the occurrence of such a resistance mechanism warrants careful antimicrobial stewardship, infection-control measures, and continued institutional microbiological surveillance.[6,7]

Analysis of clinical characteristics revealed high frequencies of potentially modifiable VAP-associated factors, including supine positioning (73.3%), enteral feeding (66.7%), and excessive sedation (53.3%). These factors may contribute to aspiration of colonized gastric and upper respiratory tract secretions and consequently facilitate the development of VAP.[1,2,14,15] Preventive strategies including head-of-bed elevation, minimization of sedation, appropriate oral care, assessment of readiness for extubation, and other evidence-based ventilator-care practices are therefore important components of VAP prevention.[7,14,15]

Limitations

First, the total cohort size (N = 150) yielded 15 VAP events; single-centre subgroup analyses of antimicrobial resistance across individual species must be interpreted cautiously. Second, clinical factors were described descriptively in the VAP cohort without formal comparative multivariable regression against the non-VAP cohort (n = 135) to establish independent predictive odds ratios.

Conclusion

Ventilator-associated pneumonia occurred in 10.0% of mechanically ventilated adult ICU patients in our cohort, driven predominantly by ESBL- and AmpC-producing Gram-negative bacilli.

Minimizing modifiable risk factors (head-of-bed elevation, sedation minimization, subglottic suctioning) combined with robust institution-specific antimicrobial stewardship policies is essential to control VAP burden and limit the selection pressure for multidrug-resistant pathogens.

References

1. Chastre J, Fagon JY. Ventilator-associated pneumonia. Am J Respir Crit Care Med. 2002;165(7):867-903.
2. Cook DJ, Walter SD, Cook RJ, et al. Incidence of and risk factors for ventilator-associated pneumonia in critically ill patients. Ann Intern Med. 1998;129(6):433-440.
3. Safdar N, Dezfulian C, Collard HR, Saint S. Clinical and economic consequences of ventilator-associated pneumonia: a systematic review. Crit Care Med. 2005;33(10):2184-2193.
4. Kollef MH. What is ventilator-associated pneumonia and why is it important? Respir Care. 2005;50(6):714-721.
5. American Thoracic Society; Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med. 2005;171(4):388-416.
6. Kalil AC, Metersky ML, Klompas M, et al. Management of adults with hospital-acquired and ventilator-associated pneumonia: 2016 clinical practice guidelines by the IDSA/ATS. Clin Infect Dis. 2016;63(5):e61-e111.
7. Klompas M, Branson R, Cawcutt K, et al. Strategies to prevent ventilator-associated pneumonia, ventilator-associated events, and nonventilator hospital- acquired pneumonia in acute-care hospitals: 2022 update. Infect Control Hosp Epidemiol. 2022;43(6):687-713.
8. Joseph NM, Sistla S, Dutta TK, Badhe AS, Rasitha D, Parija SC. Ventilator-associated pneumonia in a tertiary care hospital in India: role of multidrug-resistant pathogens. J Infect Dev Ctries. 2010;4(4):218-225.

9. Charles MP, Kali A, Easow JM, et al. Ventilator-associated pneumonia: a persistent healthcare problem in Indian intensive care units! Lung India. 2016;33(5):512-516.
10. Gupta A, Agrawal A, Mehrotra S, Singh A, Malik S, Khanna A. Incidence, risk stratification, antibiogram of pathogens isolated and clinical outcome of ventilator-associated pneumonia in a tertiary care hospital. Lung India. 2017;34(3):235-240.
11. Patro S, Sarangi G, Das P, et al. Bacteriological profile of ventilator-associated pneumonia in a tertiary care hospital. Indian J Pathol Microbiol. 2018;61(3):375-379.
12. Patil HV, Patil VC. Incidence, bacteriology, and clinical outcome of ventilator-associated pneumonia at tertiary care hospital. J Nat Sci Biol Med. 2017;8(1):46-55.
13. Chaudhury A, Rani AS, Kalawat U, Sumant S, Verma A, Venkataramana B. Antibiotic resistance and pathogen profile in ventilator-associated pneumonia in a tertiary care hospital in India. Indian J Med Res. 2016;144(3):440-446.
14. Oliveira J, Zagalo C, Cavaco-Silva P. Prevention of ventilator-associated pneumonia. Rev Port Pneumol. 2014;20(3):152-161.
15. Park SY, Lee HB. Prevention and management of ventilator-associated pneumonia. Korean J Med. 2014;86(5):537-545.