ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 06 | June - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

PROPORTION OF VENTILATOR- ASSOCIATED PNEUMONIA IN A TERTIARY CARE HOSPITAL: AN OBSERVATIONAL STUDY

Anaesthesiology

Dr. Mohit Aggarwal Senior Resident, Department of Anaesthesiology, Agartala Government Medical College (AGMC) & Gobind Ballabh Pant (GBP) Hospital, Agartala, Tripura, India. Pin: 799006

Dr. Vaskar Majumdar Professor, Department of Anaesthesiology, Agartala Government Medical College (AGMC) & Gobind Ballabh Pant (GBP) Hospital, Agartala, Tripura, India. Pin: 799006

Dr. Tapan Majumdar Professor & Head of Department, Department of Microbiology, Agartala Government Medical College (AGMC) & Gobind Ballabh Pant (GBP) Hospital, Agartala, Tripura, India. Pin: 799006

Dr. Chirasree Choudhury* Associate Professor, Department of Anaesthesiology, Agartala Government Medical College (AGMC) & Gobind Ballabh Pant (GBP) Hospital, Agartala, Tripura, India. Pin: 799006*Corresponding Author

ABSTRACT

Background: Ventilator-associated pneumonia (VAP) is one of the most important preventable causes of morbidity and mortality in critically ill patients on mechanical ventilation. Data on VAP from the North-East region of India, particularly Tripura, are scarce.**Objectives:** To determine the proportion of VAP, identify causative bacterial pathogens with antibiotic sensitivity/resistance profiles, and assess the relationship between isolated organisms and patient outcomes.**Methods:** A cross-sectional observational study was conducted in the Anaesthesia Intensive Care Unit (AICU), Agartala Government Medical College & G.B.P. Hospital, Tripura over 18 months (September 2023 – February 2025). A total of 247 mechanically ventilated patients (>48 hours) were enrolled. VAP diagnosis was established using CDC criteria. Daily endotracheal aspirate culture and antibiotic sensitivity tests were performed. Data were analysed using SPSS 27.0 and GraphPad Prism 5.**Results:** The proportion of VAP was 22.67% (56/247). Late-onset VAP predominated (55.36%) over early-onset VAP (44.64%). VAP-associated mortality was 75%, with late-onset VAP carrying higher mortality (83.8%) than early-onset (64%). The leading organisms were Klebsiella pneumoniae (30%), Acinetobacter species (18%), Pseudomonas aeruginosa (18%), and Staphylococcus aureus (16%). Multidrug resistance was widespread; colistin retained consistent activity against Gram-negatives, while vancomycin, teicoplanin, and linezolid were effective against Gram-positives.**Conclusion:** VAP in the AICU carries a high proportion and mortality, predominantly due to multidrug-resistant Gram-negative organisms. Enhanced antibiotic stewardship, infection control, and adherence to VAP prevention bundles are urgently needed.

KEYWORDS

Ventilator-associated Pneumonia, Intensive Care Unit, Multidrug Resistance, Endotracheal Aspirate, Antimicrobial Resistance, Klebsiella Pneumoniae, Acinetobacter

INTRODUCTION

Ventilator-associated pneumonia (VAP) is defined as pneumonia occurring 2 or more calendar days (≥48 hours) after endotracheal intubation or tracheostomy, caused by infectious agents not present or incubating at the time mechanical ventilation was initiated.[1] It has been recognised as one of the most important preventable causes of morbidity and mortality in critically ill patients. VAP has a cumulative incidence of 10–25%, accounts for approximately 25% of all ICU infections and 50% of antibiotic prescriptions, making it a primary focus for risk-reduction strategies.[1]

VAP rates as high as 24.5/1000 ventilator-days have been reported in cancer patients. Higher rates are consistently observed in ICUs of developing countries. VAP is associated with very high mortality, generally highest in medical ICUs and in patients infected with multidrug-resistant (MDR) pathogens.[2] Risk factors include oropharyngeal and gastric colonisation, thermal injuries, emergency intubation, reintubation, tracheostomy, bronchoscopy, nasogastric tube insertion, altered body positioning, level of consciousness, stress ulcer prophylaxis, and use of sedative agents, immunosuppressants, and antibiotics.[4]

Early-onset VAP (occurring within 4 days of ventilation) is usually attributed to antibiotic-sensitive community-acquired organisms such as *Haemophilus* and *Streptococcus* species. Late-onset VAP (after 4 days) is caused by MDR organisms including *Pseudomonas aeruginosa* and methicillin-resistant *Staphylococcus aureus* (MRSA).[3] Delay in appropriate antibiotic therapy increases mortality. VAP also prolongs ICU length of stay and increases healthcare costs.[6]

There are very few published studies on VAP from the North-East region of India, especially Tripura.[5] The present study was conducted to determine the proportion of VAP, isolate and identify causative organisms and their antibiotic sensitivity/resistance profiles, and assess the relationship between isolated organisms and patient outcomes in a tertiary care AICU in Tripura.

OBJECTIVES

1. To isolate and identify various bacterial pathogens causing VAP and determine their antibiotic sensitivity and resistance profiles.
2. To determine the relationship between outcome of VAP and the isolated organism.

MATERIALS AND METHODS

Study design and setting

This was a cross-sectional observational study conducted in the Anaesthesia Intensive Care Unit (AICU), Department of Anaesthesiology, Agartala Government Medical College & G.B.P. Hospital, West Tripura, India, over 18 months (September 2023 to February 2025).

Study population and sample size

All patients admitted to the AICU who were placed on invasive mechanical ventilation for more than 48 hours and fulfilled the inclusion criteria were enrolled. Sample size was calculated using the formula $N = Z^2PQ/L^2$, where Z = 1.96 (95% CI), P = 20% (estimated VAP proportion based on Rit et al.),[22] Q = 80, and L = 5% (allowable error), yielding a minimum sample size of 247 patients. Simple random sampling was used.

Inclusion criteria

Patients kept on mechanical ventilation for more than 48 hours in the AICU.

Exclusion criteria

(i) Patients who died or developed pneumonia within 48 hours of mechanical ventilation; (ii) those admitted with pneumonia at the time of ICU admission; (iii) patients with ARDS; (iv) patients whose attendants refused consent.

Diagnosis of VAP

VAP was diagnosed using CDC criteria,[1] evaluated daily until the patient was on mechanical ventilator support or for a maximum of 14 days, whichever was earlier. VAP was classified as: (a) early-onset VAP — developing within 4 days (96 hours) of mechanical ventilation,

and (b) late-onset VAP — developing after 4 days of mechanical ventilation.

Microbiological Sampling

Endotracheal aspirate samples were preferred over protected specimen brush (PSB) and bronchoalveolar lavage (BAL) as these are more invasive without demonstrated mortality benefit. Aspirates were collected under strict aseptic precautions using a 22-inch Romson's 12F suction catheter with a mucus extractor, introduced approximately 25 cm into the endotracheal tube. After gentle aspiration without saline instillation, 4 mL of 0.9% normal saline was injected to flush exudates into a sterile container. Samples were immediately processed by the Department of Microbiology, AGMC & G.B.P. Hospital.

Data collection

Baseline demographics (age, sex, comorbidities, prior respiratory illness), indication for mechanical ventilation, mode of airway access, ventilator settings, daily vitals (blood pressure, SpO_2, respiratory rate, temperature), tracheal secretions, total leucocyte count, PaO_2/FiO_2 ratio, chest radiograph findings, culture and sensitivity results, total ventilator days, and outcome (survival/death) were recorded.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 27.0 (SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Continuous variables are expressed as mean ± standard deviation; categorical variables as counts and percentages. Chi-square test was used for comparison of proportions. A p-value ≤0.05 was considered statistically significant.

Ethical Consideration

The study was approved by the Institutional Ethical Committee of Agartala Government Medical College & G.B.P. Hospital, Agartala, Tripura. Written informed consent was obtained from patients' attendants/guardians. All data were kept confidential and used for research purposes only.

RESULTS

Demographic and Clinical Characteristics

A total of 247 patients fulfilling the inclusion criteria were enrolled. Of these, 56 (22.67%) developed VAP. The mean age of the study population was 46.2 years; 60.32% were above 40 years. Male patients predominated (53.85% vs 46.15%). Hypertension was present in 35.63% and diabetes in 15.38% of all enrolled patients. Among VAP-positive patients, hypertension was present in 42.86% and diabetes in 19.64% (Table 1).

Table 1: Clinico-demographic Characteristics of the Study Population

Variable	Sub-group	Total (n=247)	VAP Positive (n=56)
Age	>40 years	149 (60.32%)	39 (69.64%)
	≤40 years	98 (39.68%)	17 (30.36%)
	Mean age	46.2 years	48.7 years
Sex	Male	133 (53.85%)	38 (67.86%)
	Female	114 (46.15%)	18 (32.14%)
Co-morbidities	Hypertension	88 (35.63%)	24 (42.86%)
	Diabetes mellitus	38 (15.38%)	11 (19.64%)

Proportion of VAP

Of the 247 mechanically ventilated patients, 56 (22.67%) developed VAP after ≥48 hours of mechanical ventilation. The VAP incidence per 100 patients was 22.67%.

Mortality

The overall study population mortality was 50.60% (125/247). Mortality among VAP-positive patients (75%; 42/56) was significantly higher than in VAP-negative patients (43.45%; 83/191; p=0.00006, chi-square test) (Table 2).

Table 2: Prognosis (outcome) among VAP-positive and VAP-negative patients

Outcome	VAP Present n=56 (%)	VAP Absent n=191 (%)	Total n=247 (%)
Survived	14 (25.00%)	108 (56.55%)	122 (49.40%)
Expired	42 (75.00%)	83 (43.45%)	125 (50.60%)

p=0.00006 (Chi-square test)

Early-onset vs late-onset VAP

Among the 56 VAP cases, 25 (44.64%) were early-onset and 31 (55.36%) were late-onset. Mortality was 64% in early-onset and 83.8% in late-onset VAP. Mean ventilator days were 5.38 in early and 6.26 in late VAP. Mean age of early-onset VAP patients was 45.2 years versus 51.4 years in late-onset cases (Table 3).

Table 3: Baseline Characteristics of Early-onset and Late-onset VAP

Variable	Early VAP n=25 (44.64%)	Late VAP n=31 (55.36%)
Age >40 years	15 (60.00%)	24 (77.42%)
Male sex	17 (68.00%)	21 (67.74%)
Mean age (years)	45.2	51.4
Mean day of VAP onset	3.8 days	7.6 days
Mean ventilator days	5.38 days	6.26 days
Mortality	16/25 (64.00%)	26/31 (83.80%)

Duration of mechanical ventilation

The mean duration of mechanical ventilation was 5.66 days in VAP-positive patients compared to 4.01 days in VAP-negative patients (overall mean: 4.38 days).

Microbiological profile

Of the 56 VAP-positive patients, 50 had culture-positive endotracheal aspirate. The predominant organisms were Klebsiella pneumoniae (30%), Acinetobacter species (18%), Pseudomonas aeruginosa (18%), and Staphylococcus aureus (16%). Fatality rates were 100% for Klebsiella pneumoniae, Citrobacter species, Enterobacter aerogenes, Streptococcus pyogenes, Proteus vulgaris, and Enterococcus species. Pseudomonas aeruginosa had 88.89%, Acinetobacter species 77.78%, Staphylococcus aureus 37.50%, and Escherichia coli 25.00% fatality rate (Table 4).

Table 4: Etiological agents causing VAP with proportion and fatality rate

S.No.	Causative Organism	Total (n)	Proportion (%)	Fatality Rate (%)
1	Klebsiella pneumoniae	15	30	100.00
2	Acinetobacter species	09	18	77.78
3	Pseudomonas aeruginosa	09	18	88.89
4	Staphylococcus aureus	08	16	37.50
5	Escherichia coli	04	08	25.00
6	Citrobacter species	01	02	100.00
7	Enterobacter aerogenes	01	02	100.00
8	Streptococcus pyogenes	01	02	100.00
9	Proteus vulgaris	01	02	100.00
10	Enterococcus species	01	02	100.00

Etiological agents in early-onset vs late-onset VAP

In early-onset VAP, the most common organisms were Klebsiella pneumoniae (36.36%) and Staphylococcus aureus (31.81%). In late-onset VAP, Acinetobacter species (32.14%), Klebsiella pneumoniae (25%), and Pseudomonas aeruginosa (21.42%) predominated (Table 5).

Table 5: Etiological agents causing early-onset and late-onset VAP

Etiological Agent	Early VAP n=25 (%)	Late VAP n=31 (%)
Klebsiella pneumoniae	8 (36.36%)	7 (25.00%)
Acinetobacter species	0	9 (32.14%)
Pseudomonas aeruginosa	3 (13.63%)	6 (21.42%)
Staphylococcus aureus	7 (31.81%)	1 (3.57%)
Escherichia coli	2 (9.09%)	2 (7.14%)
Citrobacter species	1 (4.54%)	0
Enterobacter aerogenes	0	1 (3.57%)
Streptococcus pyogenes	1 (4.54%)	0
Proteus vulgaris	0	1 (3.57%)
Enterococcus species	0	1 (3.57%)

Antimicrobial resistance — Gram-negative organisms

Klebsiella pneumoniae (n=15) showed 86.6% resistance to piperacillin-tazobactam and moxifloxacin, 80% to amikacin and ciprofloxacin, and 100% to cefotaxime. Acinetobacter species (n=9) showed 100% resistance to cefotaxime, meropenem, piperacillin-tazobactam, and levofloxacin; colistin sensitivity was only 22.2%. Pseudomonas aeruginosa (n=9) showed 100% resistance to

cefotaxime and piperacillin-tazobactam, and 88.8% to ciprofloxacin, meropenem, and tetracycline. Escherichia coli (n=4) showed 100% resistance to ciprofloxacin, cotrimoxazole, moxifloxacin, and piperacillin-tazobactam (Table 6).

Table 6: Antimicrobial Resistance Percentage (%) in Gram-negative Organisms Causing VAP

Organism	AK	CIP	COT	GEN	TET	CFO	CTZ	IMI	MER	PTZ	AZT	MOX	LEV	CFS	COL
Klebsiella pneumoniae (n=15)	80	80	73.3	73.3	66.6	73.3	66.6	80	73.3	86.6	73.3	86.6	73.3	60	26.6
Acinetobacter spp. (n=9)	77.7	88.8	88.8	77.7	77.7	100	88.8	66.6	100	100	77.7	88.8	100	77.7	22.2
Pseudomonas aeruginosa (n=9)	66.6	88.8	NA	66.6	88.8	100	77.7	77.7	88.8	100	66.6	77.7	77.7	66.6	22.2
E. coli (n=4)	75	100	100	75	75	75	50	50	75	100	50	100	75	75	25
Citrobacter spp. (n=1)	100	100	100	100	100	100	100	100	100	100	100	100	100	100	0
Enterobacter aerogenes (n=1)	100	100	100	100	100	100	100	0	100	100	100	100	100	100	0
Proteus vulgaris (n=1)	100	100	100	100	100	100	100	0	0	100	100	100	100	100	0

AK=Amikacin; CIP=Ciprofloxacin; COT=Cotrimoxazole; GEN=Gentamicin; TET=Tetracycline; CFO=Cefotaxime; CTZ= Ceftazidime; IMI=Imipenem; MER=Meropenem; PTZ=Piperacillin +Tazobactam; AZT=Aztreonam; MOX=Moxifloxacin; LEV= Levofloxacin; CFS=Cefoperazone+ Sulbactam; COL=Colistin; NA=Not applicable

Antimicrobial resistance — Gram-positive organisms

Staphylococcus aureus (n=8) showed 100% resistance to ciprofloxacin and 87.5% to erythromycin, cotrimoxazole, and levofloxacin, with 0% resistance to vancomycin and teicoplanin.

Streptococcus pyogenes (n=1) showed 100% resistance to erythromycin, clindamycin, ciprofloxacin, levofloxacin, and moxifloxacin. Enterococcus species (n=1) showed 100% resistance to most antibiotics except vancomycin, teicoplanin, and linezolid (Table 7).

Table 7: Antimicrobial Resistance Percentage (%) in Gram-positive Organisms Causing VAP

Organism	DOX	ERY	CLIN	GEN	CIP	COT	VAN	TEIC	LNZ	LEV	MOX
S. aureus (n=8)	37.5	87.5	75	50	100	87.5	0	0	25	87.5	75
Strep. pyogenes (n=1)	0	100	100	0	100	0	0	0	0	100	100
Enterococcus spp. (n=1)	100	100	100	100	100	100	0	0	0	100	100

DOX=Doxycycline; ERY=Erythromycin; CLIN=Clindamycin; GEN=Gentamicin; CIP=Ciprofloxacin; COT=Cotrimoxazole; VAN=Vancomycin; TEIC=Teicoplanin; LNZ=Linezolid; LEV=Levofloxacin; MOX=Moxifloxacin

DISCUSSION

This cross-sectional observational study was conducted to evaluate the proportion, microbiological profile, antimicrobial resistance, and outcomes of VAP in the AICU of a tertiary care hospital in Tripura.

Proportion of VAP

The proportion of VAP in our study was 22.67%. This is consistent with findings reported by Zirpe et al. (2015)[23] (24.94% in a neuro-ICU), Masih et al. (2016)[25] (23.54% in a retrospective MICU/CCU study), and Rit et al. (2014)[22] (21.87% in a prospective two-ICU study). Lower rates of 4.4% were reported by Vishwajith et al. (2019)[3] and 12.7% by Behera et al. (2024),[29] likely due to stricter infection control and VAP prevention bundle adherence. Higher incidences of 57.14% and 78% were reported by Ranjan et al. (2014)[34] and Deshmukh et al. (2017)[26] respectively, possibly reflecting differing diagnostic protocols or ICU environments. The variance across studies is attributable to differences in ICU settings, patient demographics, diagnostic criteria, and implementation of preventive bundles.

Early-onset vs late-onset VAP

Among 56 VAP cases, 44.64% were early-onset and 55.36% were late-onset VAP. The predominance of late-onset VAP is consistent with findings by Rit et al. (2014)[22] (60.7% late-onset), Behera et al. (2024)[29] (65.8% late-onset), Semet et al. (2023)[19] (77.1% late-onset), Masih et al. (2016)[25] (60% late-onset), Mishra et al. (2020)[36] (56% late-onset), and Gadani et al. (2010)[37] (73% late-onset). This is explained by prolonged mechanical ventilation and increasing MDR pathogen prevalence over time.

Mortality

VAP-associated mortality was 75%, significantly higher than the 43.45% mortality in VAP-negative patients (p=0.00006). Late-onset VAP mortality was 83.8% versus 64% in early-onset VAP. The elevated mortality compared to other studies — 48.33% by Ranjan et al. (2014)[34] and 31.2% by Gunalan et al. (2023)[27] — is likely attributable to MDR infections, late referrals, high comorbidity burden, and resource constraints, as highlighted by Howroyd et al. (2024).[28] The overall ICU mortality of 50.6% reflects the tertiary referral nature of the hospital and severity of illness on admission.

Duration of mechanical ventilation

VAP patients had longer mean ventilation duration (5.66 days vs 4.01 days). This aligns with Li et al. (2024)[30] (WMD 6.96 days) and Thimmaiah et al. (2024)[31] (18 vs 5.6 days), and reinforces the role of prolonged ventilation as a risk factor for VAP, as also noted by Mohanty et al. (2016)[24] and Rit et al. (2014).[22]

Microbiological profile and resistance patterns

The predominant organisms were *Klebsiella pneumoniae* (30%), *Acinetobacter* species (18%), and *Pseudomonas aeruginosa* (18%). These findings align with Kharel et al. (2021),[5] Gunalan et al. (2023),[27] Vishwajith et al. (2019),[3] and Rit et al. (2014),[22] who consistently identified these Gram-negative organisms as predominant nosocomial pathogens in ICU VAP.

Alarming antimicrobial resistance was observed with *Klebsiella pneumoniae* showing >80% resistance to amikacin, ciprofloxacin, and piperacillin-tazobactam. *Acinetobacter* spp. and *Pseudomonas aeruginosa* showed near-complete resistance to carbapenems. These findings are consistent with Mathur et al. (2014),[21] Mehrotra et al. (2023),[32] Patro et al. (2018),[33] and Zirpe et al. (2015)[23] (81.58% MDR, 12.28% XDR), underscoring the urgent need for antibiotic stewardship and surveillance.

Comorbidities and risk factors

Hypertension (42.86%) and diabetes (19.64%) were common among VAP patients, with 85% and 100% mortality respectively. These findings are supported by Othman et al. (2017)[6] and Li et al. (2024),[30] who identified diabetes and male gender as significant risk factors for VAP. Behera et al. (2024)[29] additionally reported AKI, immunosuppression, and nasogastric feeding as risk factors.

CONCLUSION

VAP remains a significant nosocomial infection in the AICU with a proportion of 22.67% and high associated mortality of 75%, particularly in late-onset VAP (83.8%). The predominant pathogens were MDR Gram-negative organisms — Klebsiella pneumoniae, Acinetobacter species, and Pseudomonas aeruginosa — with colistin retaining the most consistent activity. Among Gram-positive organisms, vancomycin, teicoplanin, and linezolid remained effective. These findings underscore the urgent need for stringent antibiotic stewardship, enhanced microbiological surveillance, strict infection control practices, and adherence to VAP prevention bundles. Future multicentre studies with larger sample sizes are warranted to validate these findings and develop optimised management guidelines.

LIMITATIONS

This was a single-centre study with a relatively small sample size, limiting generalisability. Hospital-specific practices and local epidemiology may differ from other institutions. Invasive differentiation of infection from colonisation (e.g., BAL) was not performed. ARDS patients and paediatric cases were excluded. Not all confounding factors associated with VAP outcomes were analysed.

ACKNOWLEDGEMENTS

The authors thank the faculty and staff of the Departments of Anaesthesiology and Microbiology, Agartala Government Medical

College & G.B.P. Hospital, and the entire AICU team. We are grateful to all patients and their families for participating in this study.

REFERENCES

1. AIIMS, New Delhi and Indian Council of Medical Research. VAP Surveillance Protocol. Available at: www.haisindia.com [Accessed 24 April 2023].
2. Papazian L, Klompas M, Luyt CE. Ventilator-associated pneumonia in adults: a narrative review. Intensive Care Med. 2020;46(5):888-906.
3. Vishwajith, Rao KA, Sangeetha S, Lakshminarayana SA. Ventilator associated pneumonia: An enduring hitch in intensive care units!! A study from a tertiary care center. Indian J Microbiol Res. 2019;6(3):194-197.
4. Haribhai S, Mahboobi SK. Ventilator Complications. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK560535/
5. Kharel S, Bist A, Mishra SK. Ventilator-associated pneumonia among ICU patients in WHO Southeast Asian region: A systematic review. PLoS ONE. 2021;16(3):e0247832.
6. Othman AA, Abdelazim MS. Ventilator-associated pneumonia in adult intensive care unit: prevalence and complications. Egyptian J Crit Care Med. 2017;5:61-63.
7. Center for Devices and Radiological Health. Masks and N95 Respirators. FDA; 2019.
8. Ahmed RA, Boyer TJ. Endotracheal Tube. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK539747/
9. Pujare PR, Gharatkar SR, Kerkar DP, Patil V, et al. Mechanical Ventilator. Int J Res Publ Rev. 2024;5. Available at: www.ijrpr.com
10. Ascha M, Bhattacharyya A, Ramos JA, Tonelli AR. Pulse Oximetry and Arterial Oxygen Saturation during Cardiopulmonary Exercise Testing. Med Sci Sports Exerc. 2018;50(10):1992-1997.
11. Bhutta BS, Alghoula F, Berim I. Hypoxia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK482316/
12. Castro D, Patil SM, Zubair M, Keenaghan M. Arterial Blood Gas. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025. Available at: https://www.ncbi.nlm.nih.gov/books/NBK536919/
13. Hickey SM, Sankari A, Giwa AO. Mechanical Ventilation. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK539742/
14. Mora Carpio AL, Mora JI. Assist-Control Ventilation. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing 2025. Available at: https://www.ncbi.nlm.nih.gov/books/NBK441856/
15. Wu D, Wu C, Zhang S, Zhong Y. Risk Factors of Ventilator-Associated Pneumonia in Critically Ill Patients. Front Pharmacol. 2019;10:482.
16. Goel V, Gupta S, Goel T. Ventilator-associated pneumonia: A review of the clinically relevant challenges in diagnosis and prevention. Br J Med Practitioners. 2016;9(2):a910.
17. Charles MP, Kali A, Easow JM, Joseph NM, Ravishankar M, Srinivasan S, et al. Ventilator-associated pneumonia. Australas Med J. 2014;7(8):334-344.
18. Miron M, Blaj M, Ristescu AI, Iosep G, Avadanei AN, Iosep DG, et al. Hospital-Acquired Pneumonia and Ventilator-Associated Pneumonia: A Literature Review. Microorganisms. 2024;12(1):213.
19. Semet C. The ongoing challenge of ventilator-associated pneumonia: epidemiology, prevention, and risk factors for mortality in a secondary care hospital ICU. Infect Prev Pract. 2023;5(4):100320.
20. Gutierrez JMM, Borromeo AR, Dueno AL, Paragas ED Jr, Ellasus RO, Abalos-Fabia RS, et al. Clinical epidemiology and outcomes of ventilator-associated pneumonia in critically ill adult patients: protocol for a systematic review and meta-analysis. Syst Rev. 2019;8(1):180.
21. Mathur P, Ningombam A, Soni KD, Aggrawal R, Singh KV, Samanta P, et al. Surveillance of ventilator associated pneumonia in a network of Indian hospitals using modified definitions: a pilot study. 2024. Available at: www.thelancet.com
22. Rit K, Chakraborty B, Saha R, Majumder U. Ventilator associated pneumonia in a tertiary care hospital in India: Incidence, etiology, risk factors, role of multidrug resistant pathogens. Int J Med Public Health. 2014;4:51-56.
23. Zirpe K, Kapse U, Sharma A, Kulkarni AP, Gurav S, Tiwari A, et al. Incidence, Risk Factors, Microbiology and Outcomes of VAP at an NCU in India: A Prospective Observational Study. Indian J Crit Care Med. 2025;29(4):308-313.
24. Mohanty D, Nayak MK, Raut K, Routray SS, Mishra D. Ventilator associated pneumonia in an ICU of a tertiary care hospital in India. Indian J Clin Anaesthesia. 2016;3(2):139-143.
25. Masih SM, Goel S, Singh A, Tank R, Khichi SK, Singh S. Incidence and risk factors associated with development of ventilator associated pneumonia from a tertiary care center of northern India. Int J Res Med Sci. 2016;4:1692-1697.
26. Deshmukh B, Kadam S, Thirumugam M, Rajesh K. Clinical study of ventilator-associated pneumonia in tertiary care hospital, Kolhapur, Maharashtra, India. Int J Res Med Sci. 2017;5:2207-2211.
27. Gunalan A, Sastry AS, Ramanathan V, Sistla S. Early- vs Late-onset Ventilator-associated Pneumonia in Critically Ill Adults: Comparison of Risk Factors, Outcome, and Microbial Profile. Indian J Crit Care Med. 2023;27(6):411-415.
28. Howroyd F, Chacko C, MacDuff A, Gautam N, Pouchet B, Tunnicliffe B, et al. Ventilator-associated pneumonia: pathobiological heterogeneity and diagnostic challenges. Nat Commun. 2024;15.
29. Behera AK, Guruprasad H, Shivram, Navoday, Divya, Reddy M, et al. Incidence, Risk Factors and Microbiological Profile of Ventilator Associated Pneumonia Patients in ICU in Tertiary Care Hospital. J Adv Med Pharm Sci. 2024;26(3):37-44.
30. Li W, Cai J, Ding L, Chen Y, Wang X, Xu H. Incidence and risk factors of ventilator-associated pneumonia in the intensive care unit: a systematic review and meta-analysis. J Thorac Dis. 2024;16(9):5518-5528.
31. Thimmaiah G, Pandey N, Prinja S, Jain K, Biswal M, Agarwal R, et al. Ventilator-associated pneumonia — What price does the public health system pay? Lung India. 2024;41(4):278-283.
32. Mehrotra N, Karyakarte R, Sahu R, Mukherjee B. Bacteriological Profile of Ventilator-Associated Pneumonia in Trauma Intensive Care Unit of a Tertiary Care Hospital. J Marine Med Soc. 2023;25(2):115-120.
33. Patro S, Sarangi G, Das P, Mahapatra A, Mohapatra D, Paty B, et al. Bacteriological profile of ventilator-associated pneumonia in a tertiary care hospital. Indian J Pathol Microbiol. 2018;61(3):375-379.
34. Ranjan N, Chaudhary U, Chaudhry D, Ranjan KP. Ventilator-associated pneumonia in a tertiary care intensive care unit: Analysis of incidence, risk factors and mortality. Indian J Crit Care Med. 2014;18:200-204.
35. Belay CM, Zewale TA, Amlak BT, Abebe TG, Hailu G. Incidence and Predictors of Ventilator-Associated Pneumonia Among Adult Intubated Patients in Bahir Dar Specialized Hospitals, 2021: A Retrospective Follow-Up Study. Int J Gen Med. 2022;15:8173-8182.
36. Mishra DR, Shah DS, Shah N, Prasad JN, Gupta PP, Agrawaal KK. Study of microbiological and antibiotic sensitivity pattern of ventilator associated pneumonia (VAP) in ICU of a tertiary care hospital in Nepal. J Fam Med Prim Care. 2020;9(12):6171-6176.
37. Gadani H, Vyas A, Kar AK. A study of ventilator-associated pneumonia: Incidence, outcome, risk factors and measures to be taken for prevention. Indian J Anaesth. 2010;54(6):535-540.
38. Rao S, Bhat N, Bhat ADK, Hande HM. Incidence, determinants and outcomes of ventilator associated pneumonia in medical intensive care unit: a prospective cohort study from South Western India. Int J Res Med Sci. 2021;9:1306-1312.