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Original Research Article

Bacteriological profile and antimicrobial susceptibility pattern of isolates in clinically suspected VAP cases from the sub-Himalayan region in a tertiary care hospital

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ABSTRACT

Background: Ventilator-associated pneumonia (VAP) is a major healthcare-associated infection in critically ill patients receiving mechanical ventilation. Increasing antimicrobial resistance among VAP pathogens complicates treatment and necessitates knowledge regarding bacteriological profile and antibiotic susceptibility pattern.
Methods: This descriptive cross-sectional study was conducted in the General Intensive Care Unit of Indira Gandhi Medical College, Shimla. Endotracheal aspirates from 54 clinically suspected VAP cases were collected and processed using standard microbiological techniques. Bacterial identification was performed by conventional techniques and antimicrobial susceptibility testing was carried out according to CLSI guidelines.
Results: Out of 54 samples, 51 (94.4%) were culture-positive. Gram-negative bacilli were predominant with *Klebsiella pneumoniae* being the commonest isolate (24.6%) followed by *Acinetobacter* species (19.3%), *Pseudomonas aeruginosa* (14%) and other NFGNB (14%). Multidrug-resistant organisms were frequently encountered.
Conclusions: Gram-negative bacilli, particularly *K. pneumoniae* and *Acinetobacter* species were predominant VAP pathogens. Continuous surveillance, appropriate antimicrobial stewardship, and effective infection-control measures are essential to optimize treatment and limit antimicrobial resistance.

Keywords: VAP, ETA, *Klebsiella pneumoniae*, *Acinetobacter*, *Pseudomonas aeruginosa*

INTRODUCTION

VAP is an important hospital-acquired infection (HAI) among patients in ICU receiving mechanical ventilation. It is the second most common HAI in the ICU and occurs in 6-52% patients on ventilator support.[1] Ventilator associated pneumonia (VAP) is defined as pneumonia occurring more than 48 hours after patient has been intubated and received mechanical ventilation.[2] It is characterized by inflammation of the lung parenchyma caused by organisms acquired after mechanical ventilation.[3] The International Nosocomial Infection Control Consortium found the incidence of VAP in developed countries as high as 13.6 per 1000 MV days.[4] Indian studies indicate an overall incidence rate of 9% to 58%.[5] The duration of MV (Mechanical Ventilation) increases the risk of infection. VAP primarily develops due to the presence of an Endotracheal tube or tracheostomy, which disrupts the normal anatomy and physiology of the respiratory tract, particularly the mechanisms responsible for clearing secretions such as coughing and mucociliary action. In intubated patients, a lowered level of consciousness hampers voluntary secretion clearance, causing secretions to accumulate in the oropharynx. The normal oral flora multiplies and descends into the endotracheal tube, developing an

antibiotic-resistant biofilm that extends into the lower airways. In critically ill patients, the weakened immune response to these pathogens facilitates the onset of pneumonia.[6] Risk factors for VAP include prolonged duration of mechanical ventilation, advanced age, altered sensorium, preexisting lung disease and immune suppression due to disease or medications and malnutrition. VAP can be further classified as early-onset and late-onset VAP . Early-onset VAP occurs in ventilated patients within the first four days (≤4 days) of mechanical ventilation (MV), which is usually caused by antibiotic sensitive bacteria. Early-onset VAP is caused by pathogens like *Streptococcus pneumoniae*, *Haemophilus influenzae* and methicillin-susceptible *Staphylococcus aureus* (MSSA), antibiotic sensitive enteric Gram-negative bacilli (e.g., *Escherichia coli*, *Enterobacter* species, *Proteus* species, *Serratia marcescens* and *Klebsiella pneumoniae*).

Late-onset VAP occurs in mechanically ventilated patients from the fifth day(≥5days) of MV and is caused by multidrug-resistant (MDR) pathogens such as *Pseudomonas aeruginosa*, *Acinetobacter* spp., *methicillin-resistant Staphylococcus aureus* (MRSA) and extended-spectrum beta-lactamase (ESBL) producing Enterobacteriaceae.[7] The Clinical Pulmonary Infection Score (CPIS) proposed by Pugin et al is the criteria for diagnosis and management of VAP. It is based on six variables i.e., fever, leukocytosis, tracheal secretions, oxygenation, radiographic infiltrates and semi-quantitative cultures of tracheal aspirates. Probable VAP was suspected in any patient with a CPIS >6. This study will help the clinicians to identify the causative agents of VAP and antimicrobial susceptibility pattern shall guide them regarding appropriate antimicrobial therapy.

METHODS

This cross-sectional study was conducted in the Department of Microbiology and Anaesthesia over a period of one year from November 2024 to November 2025 at Indira Gandhi Medical College (IGMC), Shimla a tertiary care Hospital of the state of Himachal Pradesh, India. In this study, 54 cases of VAP from general ICU were included, who met the criteria of VAP as per the Clinical Pulmonary Infection Score(CPIS).

Inclusion criteria

Inclusion criteria for the study were patients above the age of 18 years who were on mechanical ventilation for more the 48 hours.

Exclusion criteria

Exclusion criteria for the study were patients on mechanical ventilation for less than 48 hours . Patients who had pneumonia or other respiratory disorders like Adult Respiratory Distress Syndrome (ARDS), cystic fibrosis, cavitary lung disease, primary or metastatic lesions in the lung based on chest x-ray findings. Patients who died or developed pneumonia within 48 hours .

Specimen collection

After getting informed consent from their relatives, Endotracheal Tube aspirate (ET) was collected under strict aseptic precautions from clinically suspected patients of VAP admitted in the general ICU and sent to microbiology laboratory for further processing. The samples received were divided into two parts: one for direct gram staining and other for semi-quantitative culture

Direct gram staining and culture

The specimen was immediately transported to the microbiology laboratory and gram staining was done. Microbiological criteria for ET aspirate were ≥10 polymorphonuclear cells/low power field and ≥1 bacteria/ oil immersion field.[8] The second part of ET aspirate was cultured by semi-quantitative method based on the four-quadrant streak technique for which 10 µL of sample was inoculated on sheep blood agar and MacConkey agar. All plates were incubated at 37°C and were checked for growth after 18-24 hours. Endotracheal aspirate cultures were read semi-quantitatively by observing the growth in the four quadrants, which suggested the approximate number of colony forming units per ml (CFU/ml) of the bacteria in the specimen. The culture was graded as 1+, 2+, 3+ and 4+.

1+ = Growth in one quadrant

2+= Growth in two quadrants

3+= Growth in three quadrants

4+= Growth in four quadrants

Cultures which showed a moderate to heavy growth with 3+ or 4+ grades were considered as positive.[9] Identification of the bacterial isolates was done by various array of standard bacteriological methods and biochemical tests.

The antimicrobial susceptibility was done by the Kirby-Baeur disk diffusion technique. Accordingly susceptibility of the isolates to following antibiotics: ampicillin + sulbactam (10/10 µg), amoxicillin + clavulanic acid (20/10µg), amikacin (30 µg/disk), gentamicin (30 µg/disk), trimethoprim/sulfamethoxazole(1.25/23.75 µg/disk), cefepime (30 µg/disk), ceftriaxone (30 µg/disk), piperacillin/tazobactam (100/10 µg), ciprofloxacin (5µg), levofloxacin (5µg), tetracycline (30 µg/disk), minocycline (30 µg/disk), aztreonam (30 µg/disk), doxycycline (30 µg/disk), tobramycin (30 µg/disk), cefotaxime (30 µg/disk), imipenem (30 µg/disk), meropenem (10µg/disk), ceftazidime (30 µg/disk) was determined following the Clinical Laboratory Standard (CLSI) Guidelines 2024.[10]

RESULTS

A total of 216 patients were admitted in the general ICU, out of which 54(25%) were clinically suspected as VAP cases and endotracheal aspirates were and sent to the Department of Microbiology for further processing. VAP was predominant in age group 26-45 years followed by elderly. VAP was more common amongst male patients (74%) and (26%) were females. The most common comorbidity observed in VAP cases was trauma (26%) followed by cardiovascular disorders (18%). The major risk factor among VAP patients observed in the present study was hospitalization of >5 days (40%) followed by prior antibiotic use (33%). Gram staining revealed predominantly gram negative bacilli in 52 out of 54 samples(70.4%).

Out of 54 suspected VAP cases 51 showed significant growth in semiquantitative culture and 3 samples were sterile. Among the 51 VAP cases, 45(88.2%) were monomicrobial (one bacterial species in ETA) and 6(11.7%) were polymicrobial (two or more bacterial species in ETA), thus yielding 57 isolates as shown in Table 2. Overall, the most common isolate was *Klebsiella pneumoniae* (24.6%) followed by *Acinetobacter* species (19.3%) as shown in Figure 1. Late-onset VAP cases were more common 41(71.9%) than early-onset VAP cases 16(28.1%). Among the late onset VAP cases *Klebsiella pneumoniae* (25%) was the predominant isolate followed by *Acinetobacter* species (19%), *Pseudomonas aeruginosa* (14%) and other non-fermenter group of organisms(14%).

Table 1: Direct Gram staining of the endotracheal tube aspirate.

Gram staining	Endotracheal aspirate (n = 54)	Percentage(%)
>10PMSs/LPF and ≥ 1 bacteria/ OIF	52	96.3
<10 PMNs ≤1 bacteria/OIF+ No microorganism seen	2	3.7
Gram negative bacilli	38	70.4
Gram negative coccobacilli	8	14.8
Yeast cell	6	11.1

Table 2: Organisms isolated from Early and Late onset VAP cases.

Pathogens	Early onset VAP	Late onset VAP	Total (100%)
Klebsiella pneumoniae	2 (12.5%)	12 (29.3%)	14 (25%)
Klebsiella oxytoca	0 (0%)	2 (4.9%)	2 (4%)
***Acinetobacter* species**	5 (13.2%)	6 (14.6%)	11 (19%)
Pseudomonas aeruginosa	2 (12.5%)	6 (14.6%)	8 (14%)
NFGNB	0 (0%)	8 (19.5%)	8 (14%)
Escherichia coli	0 (0%)	2 (4.9%)	2 (4%)
Enterobacter aerogenes	4 (25%)	2 (4.9%)	6 (11%)
***Citrobacter* species**	0 (0%)	1 (2.4%)	1 (2%)
***Candida* species**	3 (18.7%)	2 (4.9%)	5 (9%)
Total	16	41	57 (100%)

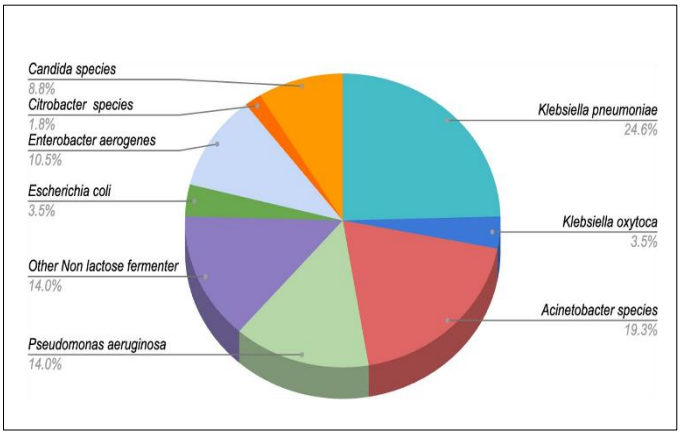


Figure 1: Distribution of pathogens isolated from VAP cases.

However, in early onset VAP cases *Acinetobacter* species (31.2%) was the commonest, followed by *Enterobacter aerogenes* (25%) and *Klebsiella pneumoniae* (12.5%). All isolates of *Klebsiella pneumoniae* were resistant to cotrimoxazole and meropenem (100%) followed by cefepime (90.1%), levofloxacin (90%) and 85.7% each for piperacillin + tazobactam and ampicillin + sulbactam as shown in Figure 2. A total of 91.3% isolates of *Klebsiella pneumoniae* were MDR. *Acinetobacter* species showed high drug resistance 100% to amikacin, tetracycline, ceftazidime, ceftriaxone, imipenem, cotrimoxazole, ciprofloxacin and gentamicin and cefotaxime followed by meropenem and levofloxacin 87.5% each as shown in Figure 3. Total 86.8% isolates of *Acinetobacter* species were MDR.

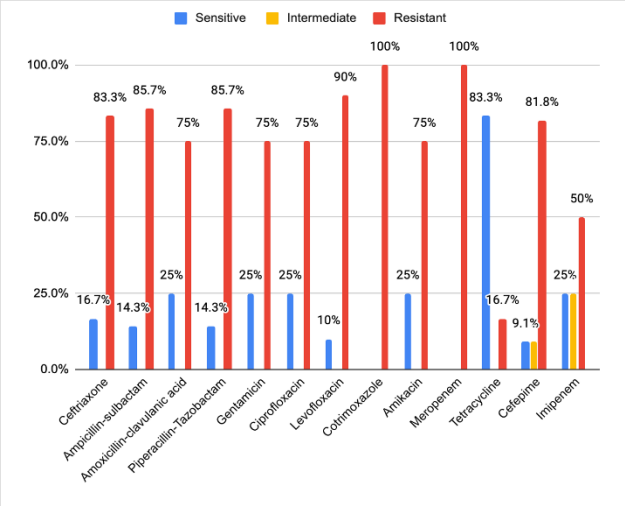


Figure 2: Antimicrobial susceptibility of *Klebsiella pneumoniae.*

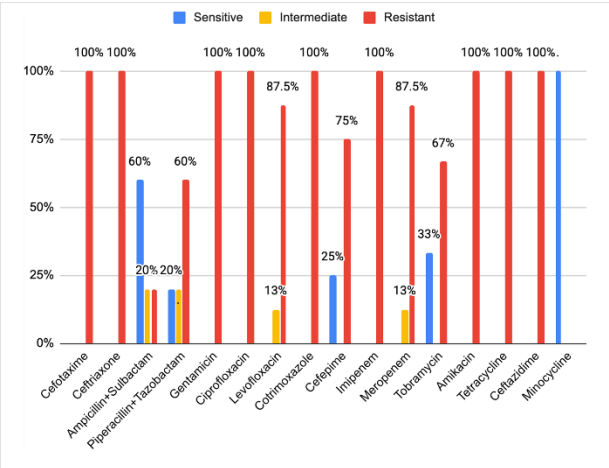


Figure 3: Antimicrobial susceptibility of *Acinetobacter* species.

All isolates of *Pseudomonas aeruginosa* were resistant to levofloxacin (100%) followed by ceftazidime (71%) as shown in Figure 4. A total of 67.8% isolates of *Pseudomonas aeruginosa* were MDR.

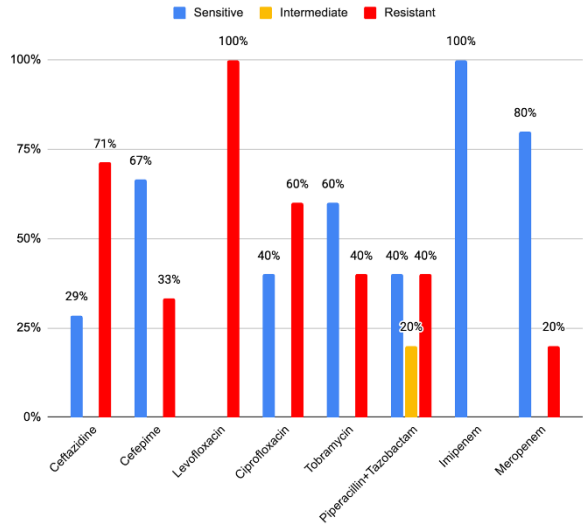


Figure 4: Antimicrobial susceptibility of *Pseudomonas aeruginosa.*

DISCUSSION

Out of the 54 endotracheal aspirates received and processed in the Department of Microbiology 51(94%) depicted significant growth on culture examination and were labelled as bacteriologically confirmed VAP, while 3 (6%) samples cultures were negative. Majority of the patients in our study belonged to the age group 26-45(31%) years followed by elderly age group (>65 years). Our findings were comparable with a study done by De et al and Goel et al who depicted that majority of the patients associated with VAP ranged between 21-40 years and 31-40 years respectively.[11,12] However a study conducted by Farag et al the highest frequency of VAP was observed in the age group of 61–80 years.[13] Age is a leading factor that plays a crucial role because with aging the immunity is reduced and elderly people become more prone to severe infections.[14] The present study showed a preponderance of male sex (76.5%) for VAP. This observation was supported by Farag et al and Goel et al, who reported 70% & 68% males in their study respectively.[13,12]

In our study, trauma (26%) was the most common comorbidity followed by cardiovascular disorders (18%). This finding is consistent with a study done by Arumugam et al, who identified trauma patients as a high-risk group for ventilator-associated pneumonia.[15] This may be attributed to emergency intubation, prolonged mechanical ventilation and impaired airway protective reflexes. In the present study, the duration of mechanical ventilation for >5 days was 40% and it was an important predisposing factor for VAP followed by prior antibiotic use in 33% patients. In our study patients admitted to the general ICU were transferred patients from other departments, so majority had received prior antibiotic therapy. Mehrotra et al also reported prior antibiotic exposure and hospitalization for >5 days as the most common risk factor for VAP.[16] Mathai et al revealed high VAP rates 37.5%

among patients who were mechanically ventilated in the ICU.[17]

A study done by Joseph et al depicted that prior antibiotic exposure and hospitalization for >5 days were independent predictors of VAP caused by multi drug-resistant pathogens.[18] Prolonged duration of hospitalization and ICU stay leads to increased antimicrobial use. The prior use of antibiotics depletes the microflora, which leads to infection with drug resistant strains of organisms. Devices like endotracheal tubes impair the host defenses and facilitate the entry of bacteria into the lung.[19] In the present study, out of total 54 endotracheal aspirates, on gram staining 52(96.3%) depicted >10 PMNs/LPF and ≥1 bacteria/OIF whereas 2 (3.7%) revealed <10 PMNs/LPF and no microorganism was seen as shown in Table 1.

Out of 54 endotracheal aspirates, 51(94.4%) were culture positive and 3(5.6%) culture were negative. This is in contrast to a study done by Patel et al who found that among 50 endotracheal aspirates, 21(40%) revealed >10 PMNs/LPF and ≥1 bacteria/ OIF and 7(14%) revealed <10 PMNs/LPF while 22 (44%) showed no microorganism.[8] Of the 54 clinically suspected VAP cases, 3 ETA samples were culture negative, but clinical and radiological evidence of VAP was present. Similar findings have been reported by Gunalan et al.[20] Out of the 51 culture positive samples, 45(88%) depicted monomicrobial growth and 6(12%) were polymicrobial.

Our results were comparable to a study conducted by Thakuria et al and Farag et al in which monomicrobial growth was 94.3% and 94% respectively.[21,13] However polymicrobial growth was 5.7% and 6% respectively. In the present study, the most common isolate was *Klebsiella pneumoniae* 24.6% followed by *Acinetobacter* species 19.3%, *Pseudomonas aeruginosa* 14%, other non-fermenter group of organisms (NFGNB) 14%, *Enterobacter aerogenes* 10.5% and *Citrobacter* species 1.8% and *candida* species 8.8%. This is consistent with findings of Mehrotra et al who reported *Klebsiella pneumoniae* (44%) as the predominant isolate followed by *Acinetobacter* species (17.8%).[16] This is in contrast to a study conducted by Taleie et al where Gram positive cocci particularly *Staphylococcus aureus* was the predominant isolate (58.7%).[22] Knowledge of variations in the microbial etiology of VAP across different ICU settings is crucial, as it aids clinicians in selecting appropriate antimicrobial therapy and helps to ensure effective management of VAP cases.

In our study 29.6% were early-onset VAP cases, while 70.4% were late-onset VAP cases. This is in concordance with a study done by Patro et al and Sharma et al where 32% and 13.8% were early-onset VAP cases respectively, however late-onset VAP cases were 68% and 86.2% respectively.[23,24] In the present study among early-onset VAP cases *Acinetobacter* species (31.2%) was the most common isolate followed by *Enterobacter aerogenes* (25%), *Pseudomonas aeruginosa* and *Klebsiella* pneumoniae 12.5% each. Our findings corroborate with the study done by Khalil et al who reported *Acinetobacter* species (43.3%) as predominant pathogen in early-onset VAP cases followed by *Enterobacter aerogenes* (40%).[14] Contrary to this Patro et al reported *Klebsiella pneumoniae* (26.7%) as the most common isolate in early-onset VAP followed by *Enterobacter* species (13.3%) and Pseudomonas aeruginosa (6.7%).[23]

Similarly, Farag et al reported *Klebsiella pneumoniae* (43.4%) as the predominant organism in early-onset VAP.[13] In our study in late-onset VAP cases, *Klebsiella pneumoniae* (25%) was the predominant isolate followed by *Acinetobacter* species (19%), *Pseudomonas aeruginosa* and other non-fermenter group of organisms 14% each. This is in concordance to a study done by Farag et al who reported *Klebsiella pneumoniae* 26.7%, *Pseudomonas aeruginosa* 26.7% and *Acinetobacter* species 6.7% in late-onset VAP cases.[13] However Patro et al reported *Acinetobacter* species (31.2%) as the commonest isolate followed by *Klebsiella pneumoniae* 15.6%.[24] The distribution of etiological agents is variable in different ICUs depending up on patient demographics, duration of hospitalization, length of ICU stays and hospital antibiotic policy. Overall *Klebsiella pneumoniae* was the predominant isolate (24.6%) in our study.

All isolates of *Klebsiella pneumoniae* were resistant to meropenem (100%), followed by cefepime (90.1%), levofloxacin (90%), ampicillin + sulbactam (85.7%) and amikacin (75%). A similar resistance pattern was observed in the study done by Set R et al where all isolates were resistant to cefepime and ampicillin + sulbactam (85.7%).[25] On the contrary Patro et al reported higher resistance to amikacin (60%), but lower resistance to meropenem (40%).[23] The inter study differences may be attributed to institutional antibiotic policies, infection control practices and differences in the study design. Additionally, 91.3% of *Klebsiella pneumoniae* isolates in our study were multi drug resistant (MDR). Similarly high MDR rates were reported by Ramadan et al (90.4%), whereas Lidhiya et al documented a lower prevalence of 38%.[26,27] *Acinetobacter* species was the second most common isolate (19.3%) in our study. *Acinetobacter* species exhibited high resistance rate to multiple antibiotics. Complete resistance was seen for gentamicin, ceftazidime, imipenem, ciprofloxacin, amikacin, followed by levofloxacin and meropenem (87.5% each) and tobramycin (66.7%). A similar resistance pattern was observed in a study conducted by Mehrotra et al in which maximum resistance (100%) was reported to ceftazidime, imipenem and ciprofloxacin, followed by levofloxacin (89.5%), amikacin (88.9%) and gentamicin (85.2%).[16] Additionally, Goel et al and Erdem et al reported higher resistance rates to ceftazidime, 100% and 90% respectively.[12,28]

In the present study 86.8% isolates of *Acinetobacter* species were MDR, consistent with findings reported by Abdollahi et al and Gupta et al.[29,30] However, Lidhiya et al

and Yadav et al have documented lower MDR resistance rate 26% and 37.5% respectively.[27,31] The high level of resistance observed in the present study suggests the possible presence of multi drug-resistant (MDR) *Acinetobacter* species. In ICU setting, particularly among mechanically ventilated patients, risk factors such as prolonged hospitalization, prior exposure to broad-spectrum antibiotics, invasive procedures and biofilm formation on endotracheal tubes contribute significantly to the emergence and persistence of resistant strains.

Overall, the findings highlight the alarming burden of drug-resistant *Acinetobacter* species in VAP cases. The preserved activity of minocycline offers a potential therapeutic option, however widespread resistance to carbapenems and aminoglycosides is of great concern. Continuous antimicrobial surveillance, strict adherence to infection control measures, adopting care bundle approach and implementation of robust antibiotic stewardship programs are essential to limit further spread of resistance.

In the present study all isolates of *Pseudomonas aeruginosa* were resistant to levofloxacin (100%), followed by ceftazidime (71%), ciprofloxacin (60%), piperacillin + tazobactam (40%) and cefepime (33.3%). These findings are in concordance with Mehrotra et al who reported high resistance rates to levofloxacin (92%) and ciprofloxacin (76.9%).[16] Rashid et al, also documented maximum resistance to levofloxacin (82.8%), followed by ciprofloxacin (75.4%) and ceftazidime (51.5%).[32] Multi-drug resistance was observed in 67.8% of *Pseudomonas aeruginosa* isolates in the present study, in concordance with a study done by De et al and Zirpe et al.[11,33] However, lower MDR rates were documented by Golia et al and Kapaganty et al.[34,35]

The increase in resistance to fluoroquinolone could be explained by wide spread empirical use, selective pressure and the emergence of chromosomal mutations and DNA gyrase and topoisomerase IV gene. Sharma et al similarly documented higher resistance rate to ciprofloxacin and ceftazidime, further supporting the declining the utility of these agents in empirical therapy for *Pseudomonas aeruginosa* infections.[24] Amongst non-fermenter group of organisms (NFGNB), in the present study all isolates were resistant to ceftriaxone(100%) and aztreonam (100%).

These findings are consistent with Mehrotra et al who reported 100% resistance to aztreonam.[16] In contrast Chaudhury et al reported a lower resistance rate of azteronam and ceftriaxone 51.9% and 56.7% respectively.[36] In the present study isolates of *Enterobacter aerogenes* showed resistant to gentamicin, ciprofloxacin and amikacin which is in concordance to a study conducted by Sharma et al and Mehrotra et al.[24,16] On the contrary Kapaganty et al reported 100 % sensitivity to amikacin and gentamicin.[35]

In our study all isolates of *Escherichia coli* were totally resistant to ampicillin, amoxicillin + clavulanic acid and levofloxacin. This is in concordance with a study conducted by Umara et al, who reported resistance to ampicillin (100%) and amoxicillin + clavulanic acid (98.2%).[32]

A limitation of our study was the unavailability of VITEK-based identification and molecular testing of the bacterial isolates. Furthermore, additional tests for the detection of multi drug resistance mechanisms could not be performed.

CONCLUSION

Ventilator-associated pneumonia (VAP) remains an important healthcare-associated infection associated with increased morbidity, mortality, hospital stay and antimicrobial use. In this study, *Klebsiella pneumonia* was the major etiological agent followed by *Acinetobacter* spp. and *Pseudomonas aeruginosa*. A high prevalence of multidrug-resistant isolates was observed, emphasizing the growing challenge of antimicrobial resistance in ICU settings. No distinct microbiological difference was observed between early and late-onset VAP, suggesting limited utility of conventional time-based classification for empirical therapy. Continuous microbiological surveillance, local antibiograms, antimicrobial stewardship and strict infection-prevention practices are essential to improve outcomes and reduce VAP burden.

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