Abstract

Background: Nosocomial pneumonia poses great challenge to an intensivist. Detailed information about hospital-acquired pneumonia (HAP) and ventilator-acquired pneumonia (VAP) is crucial for prevention and optimal management, thus improving quality Intensive Care Unit (ICU) care. Hence, we aimed to study the current trend of nosocomial pneumonia in ICU. Materials and Methods: It was a prospective observational cohort study, conducted in the ICU of a tertiary care teaching public hospital over a period of 18 months. We studied clinical profile and outcome of 120 adult patients who developed VAP/HAP during the study period. We also analyzed the causative organisms, antibiotic sensitivity, and resistance pattern in these patients. Results: Out of 120 patients, 29 patients were HAP and 91 patients were VAP. Mortality was 60% (72), and development of VAP and requirement of mechanical ventilation showed significant association with mortality ($P < 0.00001$). Most common organism causing HAP was Staphylococcus aureus (43.4%) and VAP was Klebsiella pneumoniae (49%). Maximum antibiotic sensitivity was found to piperacillin + tazobactam (58.8%), followed by imipenem (49.5%) and meropenem (41.8%), whereas maximum antibiotic resistance was found to cefepime (95.1%), followed by ceftazidime and amoxicillin (91.2%). Conclusion: Nosocomial pneumonia showed high incidence (17.44%) and mortality (60%). Common organisms identified were S. aureus and K. pneumoniae. Resistance was high for commonly used antibiotics and high antibiotic sensitivity for piperacillin + tazobactam and carbapenem.

Keywords: Hospital-acquired pneumonia, mortality, nosocomial pneumonia, ventilator-acquired pneumonia

Résumé

Contexte: La pneumonie nosocomiale pose un grand défi à un intensiviste. Des informations détaillées sur la pneumonie acquise dans les hôpitaux (HAP) et la pneumonie acquise par le ventilateur (VAP) sont essentielles pour la prévention et la gestion optimale, améliorant ainsi les soins de soins intensifs de qualité (UTI). Par conséquent, nous avons cherché à étudier la tendance actuelle de la pneumonie nosocomiale en UTI. Matériaux et méthodes: il s’agissait d’une étude de cohorte observationnelle prospective menée dans l’UTI d’un hôpital public d’enseignement tertiaire sur une période de 18 mois. Nous avons étudié le profil clinique et le résultat de 120 patients adultes qui ont développé le VAP / HAP pendant la période d’étude. Nous avons également analysé les organismes responsables, la sensibilité aux antibiotiques et le modèle de résistance chez ces patients. Résultats: Sur 120 patients, 29 patients étaient HAP et 91 patients étaient VAP. La mortalité était de 60% (72), et le développement du VAP et l’exigence de ventilation mécanique ont montré une association significative avec la mortalité ($P < 0.00001$). L’organisme le plus fréquent causant HAP était Staphylococcus aureus (43,4%) et VAP était Klebsiella pneumoniae (49%). Une sensibilité antibiotique maximale a été observée chez la pipéracilline + tazobactam (58,8%), suivie de l’imipénème (49,5%) et du méropénème (41,8%), alors que la résistance antibiotique maximale a été observée à cefépime (95,1%), suivie de ceftazidime et de l’amoxicilline (91,2%). Conclusion: La pneumonie nosocomiale a montré une incidence élevée (17,44%) et la mortalité (60%). Les organismes communs identifiés étaient S. aureus et K. pneumoniae. La résistance était haute pour les antibiotiques couramment utilisés et la sensibilité antibiotique élevée pour la pipéracilline + tazobactam et le carbapénème.

Keywords: Pneumonie nosocomiale, mortalité, pneumonie nosocomiale, pneumonie acquise par le ventilateur

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Introduction

The term nosocomial pneumonia broadly covers all infections occurring 48 h or more after hospital admission excluding any infection incubating at the time of admission. It includes hospital-acquired pneumonia (HAP) occurring in nonintubated patients. Ventilator-acquired pneumonia (VAP) is a pulmonary infection that acquired after 48 h of endotracheal intubation and invasive mechanical ventilation (MV). The HAP and VAP can be early-onset (within 2–4 days) and late-onset (>4 days). It has been shown that nosocomial pneumonia acquired in Intensive Care Unit (ICU) prolongs the hospital stay, duration of MV, morbidity, and mortality and thus considerably increases health-care cost.\(^1\)\(^-\)\(^5\) The incidence of nosocomial pneumonia in ICU ranges from 9% to 58% with variation relating to different ICU and mortality ranges from 30% to 70%.\(^1\)\(^-\)\(^6\)\(^-\)\(^8\)

Management of nosocomial pneumonia has been proved to be difficult. The clinical presentation and organism are different in different setups. Hence, there is a need for early diagnosis and development of effective prophylactic and therapeutic strategies aiming to decrease mortality rates and also to optimize the use of antimicrobial agents.\(^1\)\(^-\)\(^10\) For rational utilization of antimicrobial agents, local microbial flora causing VAP needs to be studied.

Detailed information about trend of HAP and VAP in our patient subsets is crucial to the advance of more effective preventive measures and optimal management, thus improving quality ICU care. Hence, we decided to study nosocomial pneumonia in our tertiary care teaching public hospital ICU patient subsets. The primary objective of the study was to determine the clinical profile and outcome of nosocomial pneumonia in Medical ICU (MICU). We also analyzed the causative organisms of HAP and VAP and the antibiotic sensitivity and resistance pattern in these patients and its relation with the outcome.

Materials and Methods

It was a prospective observational cohort study, conducted after approval from Institutional Ethics Committee and valid, written, informed consent from patients. Sample size was calculated from http://epitools.ausvet.com.au using the formula \(n = (Z^2 \times P \times (1 − P))/e^2\) where \(Z\) is value from standard normal distribution corresponding to desired confidence levels (\(Z = 1.96\) for 95% confidence interval [CI]), \(P\) is expected true proportion, \(e\) is desired precision (half desired CI width), and for small populations, \(n\) can be adjusted so that \(n\ (adj) = (N \times n)/(N + n)\). As per published data, considering local mortality as 10% and population size of 400 patients approximately admitted to MICU as per medical records of our hospital, where estimated true proportion was 0.10 and population size for finite population as 400, the estimated sample size for the current study was 104 and the power of the study was 80.\(^9\)\(^-\)\(^10\) Inclusion criteria to our study were all consecutive adult patients (age 18–90 years) developing pneumonia after 48 h of admission to MICU. HAP in nonintubated patients after 48 h of admission to ICU and VAP in intubated patients after 48 h of endotracheal intubation in ICU were studied. For confirmatory diagnosis of HAP and VAP, clinical diagnostic criteria and modified clinical pulmonary infection score were used as reference standard.\(^1\)\(^,\)\(^8\)\(^-\)\(^10\) The HAP and VAP were classified into early-onset type (within 2–4 days) and late-onset type (>4 days).\(^1\)\(^2\)\(^,\)\(^1\)\(^3\)

Patients who refused consent, who had already developed pneumonia before admission to MICU, and also patients with other causes of radiological infiltrate such as pulmonary hemorrhage, pulmonary embolism, atelectasis, congestive cardiac failure, and acute respiratory distress syndrome by computed tomography scan and other diagnostic modalities were excluded from the study.

During 18-month study period, 120 patients developed HAP or VAP were included in the study, however, the estimated sample size for the current study was 104. Detailed demographic, vital parameters were noted in all patients and were monitored until either discharge from ICU or death. Patients were managed in ICU according to the standard routine protocol.\(^1\)\(^4\) In all suspected patients of nosocomial pneumonia, sputum and tracheal aspirate specimen were collected aseptically and sent to microbiology for culture and antibiotic susceptibility test which were done by standard methods and criteria.\(^1\)\(^-\)\(^4\) Necessary investigations and imaging were done, and standard supportive management strategy was started. Initially, empirical antimicrobial therapy was started, and after culture sensitivity report, specific target antibiotic administered. APACHE II score was calculated at the time of admission.

Study design

It was a prospective observational cohort study and was carried out in the MICU of a tertiary care teaching public hospital in western India over a period of 18 months.

Statistical analysis

Quantitative data were represented in form of frequency and percentage. Data were represented using mean ± standard deviation (SD) and median, wherever applicable. \(P\) value was taken as significant when <0.05 and 95% CI of that fraction. Association between qualitative variables was assessed by Chi-square test with continuity correction for all 2 × 2 tables and with or without continuity correction in rest and Fisher’s exact test for all 2 × 2 tables, where \(P\) value of Chi-square
test was not valid due to small counts. Analysis of qualitative variable with two subgroups was done using unpaired t-test. Predictiveness of factors for mortality by various independent variables was assessed using binary logistic regression analysis. Results were graphically represented where necessary. SPSS version 19 (IBM SPSS Statistics) Armonk, NY: IBM Corp. USA was used for most analysis and MS Excel for graphs.

RESULTS

During the study period, out of total 688 admissions in MICU, 120 patients developed nosocomial pneumonia and thus incidence was 17.44%. The mean age ± SD for age distribution is 39.82 ± 47.83 and maximum patients belonged to the age group 19–30 years (42.5%). Out of 120 patients, 59 (49%) were male and 61 (51%) were female.

We studied 120 patients of nosocomial pneumonia; out of them, 29 patients were not mechanically ventilated and were labeled as HAP, among which 23 (79%) were early-onset HAP and 6 (21%) were late-onset HAP. Out of 120 patients of nosocomial pneumonia, 91 were mechanically ventilated and were labeled as VAP, among which 54 (59.3%) were early-onset VAP and 37 (40.7%) were late-onset VAP.

Risk factors associated with nosocomial pneumonia are summarized in Table 1. Among them, significant association with mortality was found with requirement of MV (75.8%) (P < 0.00001); also, very high mortality was noted in chronic obstructive pulmonary disease (COPD) patients (83.3%). The mean APACHE II score at time of admission was 16.85. We found that as APACHE II score increased, mortality also increased significantly and can be used as effective tool to determine outcome and accordingly modify treatment strategy in these patients. Binary logistic regression analysis demonstrated that length of stay in MICU (P = 0.023), need of MV (P = 0.00002), APACHE II score at time of admission (P = 0.000015), and Acinetobacter baumannii extended-spectrum beta-lactamases (P = 0.002) predicted worst outcome.

Out of total 120 patients of nosocomial pneumonia, 72 (60%) died and 48 (40%) patients survived. Out of 91 patients of VAP, 69 (76%) patients died and 22 (24%) patients survived, whereas out of 29 patients of HAP, 3 (10%) patients died and 26 (90%) patients survived. This shows significant association between development of VAP and mortality (P < 0.05).

Organisms isolated in early- and late-onset HAP and VAP are depicted in Tables 2 and 3. Most common organism causing early-onset HAP is Staphylococcus aureus 10 (43.4%) and VAP is Klebsiella pneumoniae 26 (49%). Most common organism causing late-onset HAP is K. pneumoniae 4 (57%) while most common organism causing late-onset VAP is K. pneumoniae 46 (45%).

Antibiotic sensitivity and resistance for individual organism is summarized in Table 4. K. pneumoniae showed maximum antibiotic sensitivity to piperacillin + tazobactam (60.3%), followed by imipenem (47.4%) and meropenem (39.7%) while maximum resistance to ceftazidime (100%), amoxicillin + clavulanic acid (100%), and amoxicillin (100%). Acinetobacter showed maximum antibiotic sensitivity to piperacillin + tazobactam (53.3%) while maximum resistance to ceftazidime (100%), cefepime (100%), and amoxicillin (93.3%). Escherichia coli showed maximum antibiotic sensitivity to imipenem (71.4%) while maximum resistance to cefepime (92.9%), amoxicillin (92.9%), and amoxicillin + clavulanic acid (92.9%). Pseudomonas aeruginosa showed maximum antibiotic sensitivity to piperacillin + tazobactam (55.3%), followed by imipenem (44.7%) and meropenem (39.5%), while maximum resistance to amoxicillin (100%) and cefepime (97.4%). S. aureus showed maximum antibiotic sensitivity to ciprofloxacin (83.3%) and methicillin (83.3%), followed by vancomycin (77.8%), while maximum resistance to cefepime (88.9%), followed by ceftazidime (72.2%). Streptococcus pneumoniae showed maximum antibiotic sensitivity to ciprofloxacin (100%), followed by amoxicillin + clavulanic acid, vancomycin, methicillin, amikacin, and piperacillin + tazobactam (80%), while maximum resistance to cefepime (80%), followed by meropenem, imipenem, ceftiraxone, and ceftazidime (40%).

Overall antibiotic sensitivity and resistance for antibiotics is summarized in Table 5. Thus, maximum antibiotic sensitivity was found to piperacillin + tazobactam (58.8%), followed by imipenem (49.5%) and meropenem (41.8%), whereas

| Table 1: Risk factors associated with nosocomial pneumonia |
|----------------------------------------------------------|
| **Risk factor** | **Number of patients** | **Mortality (%)** | **P** |
|-----------------|------------------------|------------------|------|
| Age>60 (years)  | 15 (71.4)              | 6 (28.6)         | 71.4 | 0.239 |
| Smoking         | 11 (57.9)              | 8 (42.1)         | 57.9 | 0.838 |
| Requirement of mechanical ventilation | 69 (75.8) | 22 (24.2) | 75.80 | <0.00001 |
| COPD            | 5 (83.3)               | 1 (16.7)         | 83.30 | 0.474 |
| DM              | 17 (65.4)              | 9 (34.6)         | 65.40 | 0.652 |
| CKD             | 0                      | 4 (100)          | 0    | 0.24  |
| Cardiac diseases| 8 (53.3)               | 7 (46.7)         | 53.30 | 0.573 |
| Surgical intervention | 1 (50) | 1(50) | 50.00 | 1 |

COPD=Chronic obstructive airway disease, DM=Diabetes mellitus, CKD=Chronic kidney disease
Table 2: Organisms isolated in early-onset hospital-acquired pneumonia and ventilator-acquired pneumonia

| Organisms isolated | Type of pneumonia | Hospital-acquired pneumonia (%) | Ventilator-acquired pneumonia (%) |
|--------------------|-------------------|---------------------------------|----------------------------------|
| Klebsiella pneumoniae | 4 (17.3) | 26 (49) |
| Staphylococcus aureus | 10 (43.4) | 5 (9.43) |
| Pseudomonas | 4 (17.3) | 8 (15.09) |
| Mixed | 0 | 6 (11.3) |
| Escherichia coli | 2 (8.6) | 4 (7.5) |
| Streplococcus pneumoniae | 3 (13.04) | 1 (1.8) |
| Acinetobacter | 0 | 3 (5.6) |
| Total | 23 (100) | 53 (100) |

Table 3: Organisms isolated in late-onset hospital-acquired pneumonia and ventilator-acquired pneumonia

| Organisms isolated | Type of pneumonia | Hospital-acquired pneumonia (%) | Ventilator-acquired pneumonia (%) |
|--------------------|-------------------|---------------------------------|----------------------------------|
| Klebsiella pneumoniae | 4 (57.1) | 46 (45.09) |
| Pseudomonas | 3 (42.8) | 23 (22.54) |
| Acinetobacter | 0 | 12 (11.7) |
| Escherichia coli | 0 | 9 (8.8) |
| Mixed | 0 | 6 (5.8) |
| Staphylococcus aureus | 0 | 4 (3.9) |
| Streplococcus pneumoniae | 0 | 2 (1.9) |
| Total | 7 (100) | 102 (100) |

maximum antibiotic resistance was found to cefepime (95.1%), followed by ceftazidime and amoxicillin (91.2%).

**DISCUSSION**

The incidence of nosocomial pneumonia in the present study was 17.44%. The reported incidence in literature varies from 9% to 58%.[1-5,13] In large-scale European Prevalence of Infection in Intensive Care (EPIC) study, the overall nosocomial pneumonia prevalence was 9.6%.[15] Higher incidences in Indian studies have been mentioned.[1,3,13] This broad variation makes interstudy comparison complicated. Variation in incidence may be due to different patient subsets in different ICUs, different diagnostic techniques, standard management protocol of different ICU, number of patients in each study, etc. Furthermore, high workload and inadequate nursing staff in public hospital may compromise the quality care increasing health care-associated infections.[14] Incidence varies with type of ICU also as trauma and surgical ICUs have higher rates of VAP compared to the MICUs.[13]

The mean age of the present study population was 39.82 years, and equal distribution among both sexes was found. Literature mentions similar age distribution; however, they found predominance of male population.[1-5,13]

The overall mortality for nosocomial pneumonia in this study was 60%. Furthermore, mortality was significantly high in VAP (76%) and more so in late-onset VAP. The mortality rate for VAP in studies done by Ranjan et al. (48.3%), Joseph et al. (16.2%), Fagon et al. (54.2%), etc., was varying, however, Chastre and Fagon demonstrated that it can range from 24% to 50% and can reach 76% in some specific settings or when lung infection is caused by high-risk pathogens.[1-6,16-18]

Thus, HAP and early-onset VAP had a good prognosis in terms of mortality. High mortality in late-onset VAP may be due to intubation, MV, and prolonged hospital stay. The common risk factors for development of nosocomial pneumonia identified in the present study were advanced age, smoking, need for MV, diabetes mellitus, COPD, chronic renal failure, and surgical intervention. These factors were also associated with high mortality, particularly MV (75.8%) and COPD (83.3%) [Table 1]. As this study was carried out in a tertiary care referral center, the mortality was high.

Thus, multiple risk factors and comorbidities increase the risk of nosocomial pneumonia in ICU, especially MV, longer duration of stay in ICU, and underlying chronic illness like COPD. Fagon et al. documented that the risk of infection increases with duration of hospital stay which is in accordance to our study as mortality is higher in late-onset HAP and late-onset ventilator-associated pneumonia.[16] The EPIC study reported that medical admission, admission after emergency surgery or trauma, the presence of COPD, underlying chronic illness, older age, MV, and higher APACHE II score were found to be independently associated with a higher risk of infection.[14]

Trauma was mentioned as most common underlying condition by Ranjan et al.[1] However, the current study was done at MICU and did not include any trauma patient. Rit et al. described MV and reintubation as significant risk factors.[2] Furthermore, emergency intubation in obtunded patients may increase risk of aspiration.[15]

The findings in the current study and other recent studies emphasize on avoiding unnecessary prolonged hospitalization, intubation, and MV of the patient. Considering noninvasive ventilation (NIV) whenever possible, an early and appropriate ventilator weaning should be the protocol.[9-23]

In our study, the most common organism causing early-onset HAP was *S. aureus* while most common organism causing late-onset HAP and early- and late-onset VAP was *K. pneumoniae*, followed by *Pseudomonas* and *Acinetobacter*. Other studies also showed *K. pneumoniae, Acinetobacter* spp., *P. aeruginosa,* and multidrug-resistant (MDR) pathogens as the most common organisms isolated in the patients with *HAP*.[5,12,15] Ranjan et al. in a recent study found that *Acinetobacter* spp., *P. aeruginosa,* and *K. pneumoniae* were predominant isolates.[11] Gadani et al. mention that the order of prevalence of organism was in the order of *Pseudomonas* and *Klebsiella*, followed by methicillin-resistant...
### Table 4: Antibiotic sensitivity and resistance for individual organism

| Antibiotics | Acinetobacter, n (%) | Escherichia coli, n (%) | Klebsiella pneumoniae, n (%) | Mixed, n (%) | Pseudomonas aeruginosa, n (%) | Staphylococcus aureus, n (%) | Streptococcus pneumoniae, n (%) |
|-------------|----------------------|------------------------|-----------------------------|--------------|-------------------------------|-------------------------------|-------------------------------|
| Amoxicillin | S 1 (6.7)            | 1 (7.1)                | 2 (2.6)                     | 0            | 0                             | 9 (50.0)                     | 3 (60.0)                     |
|             | R 14 (93.3)          | 13 (92.9)              | 78 (100)                    | 12 (100)     | 38 (100)                      | 9 (50.0)                     | 2 (40.0)                     |
| Amoxicillin + clavulanic acid | S 2 (13.3) | 1 (7.1) | 2 (2.6) | 0 | 2 (5.3) | 13 (72.2) | 4 (80.0) |
| Methicillin | S 3 (20.0)           | 2 (14.3)               | 5 (6.4)                     | 0            | 2 (5.3)                       | 15 (83.3)                    | 4 (80.0)                     |
| Vancomycin  | S 4 (26.7)           | 4 (28.6)               | 9 (11.5)                    | 0            | 4 (10.5)                      | 14 (77.8)                    | 4 (80.0)                     |
| Meropenem   | S 4 (26.7)           | 7 (50.0)               | 31 (39.7)                   | 4 (33.3)     | 15 (39.5)                     | 12 (66.7)                    | 3 (60.0)                     |
|             | R 10 (66.7)          | 7 (50.0)               | 44 (56.4)                   | 6 (50.0)     | 20 (52.6)                     | 6 (33.3)                     | 2 (40.0)                     |
| I/S         | 1 (6.7)              | 1 (7.1)                | 5 (6.4)                     | 2 (16.7)     | 3 (7.9)                       | 0                            | 0                            |
| Imipenem    | S 7 (46.7)           | 10 (71.4)              | 37 (47.4)                   | 5 (41.7)     | 17 (44.7)                     | 11 (61.1)                    | 3 (60.0)                     |
|             | R 7 (46.7)           | 4 (28.6)               | 35 (44.9)                   | 5 (41.7)     | 19 (50.0)                     | 7 (38.9)                     | 2 (40.0)                     |
| I/S         | 1 (6.7)              | 3 (21.4)               | 8 (10.3)                    | 2 (16.7)     | 2 (5.3)                       | 0                            | 0                            |
| Ceftriaxone | S 2 (13.3)           | 6 (42.9)               | 13 (16.7)                   | 1 (8.3)      | 6 (15.8)                      | 10 (55.6)                    | 3 (60.0)                     |
|             | R 13 (86.7)          | 8 (57.1)               | 67 (85.9)                   | 11 (91.7)    | 32 (84.2)                     | 8 (44.4)                     | 2 (40.0)                     |
| Ceftazidine | S 0                  | 2 (14.3)               | 2 (2.6)                     | 1 (8.3)      | 3 (7.9)                       | 5 (27.8)                     | 3 (60.0)                     |
|             | R 15 (100)           | 12 (85.7)              | 78 (100)                    | 11 (91.7)    | 35 (92.1)                     | 13 (72.2)                    | 2 (40.0)                     |
| Cefepime    | S 0                  | 1 (7.1)                | 3 (3.8)                     | 1 (8.3)      | 1 (2.6)                       | 2 (11.1)                     | 1 (20.0)                     |
|             | R 15 (100)           | 13 (92.9)              | 77 (98.7)                   | 11 (91.7)    | 37 (97.4)                     | 16 (88.9)                    | 4 (80.0)                     |
| Amikacin    | S 6 (40.0)           | 6 (42.9)               | 16 (20.5)                   | 3 (25.0)     | 6 (15.8)                      | 12 (66.7)                    | 4 (80.0)                     |
|             | R 9 (60.0)           | 8 (57.1)               | 64 (82.1)                   | 9 (75.0)     | 32 (84.2)                     | 6 (33.3)                     | 1 (20.0)                     |
| Ciprofloxacin | S 4 (26.7) | 5 (35.7) | 14 (17.9) | 2 (16.7) | 3 (7.9) | 15 (83.3) | 5 (100) |
|             | R 11 (73.3)          | 9 (64.3)               | 66 (84.6)                   | 10 (83.3)    | 35 (92.1)                     | 3 (16.7)                     | 0 (0.0)                      |
| Piperacillin + tazobactam | S 8 (53.3) | 7 (50.0) | 47 (60.3) | 7 (58.3) | 21 (55.3) | 13 (72.2) | 4 (80.0) |
|             | R 7 (46.7)           | 7 (50.0)               | 31 (39.7)                   | 5 (41.7)     | 17 (44.7)                     | 5 (27.8)                     | 1 (20.0)                     |
| Total isolates | 15 (100) | 14 (100) | 78 (100) | 12 (100) | 38 (100) | 18 (100) | 5 (100) |

S=Sensitivity, R=Resistance, I/S=Intermediate sensitivity

### Table 5: Overall antibiotic sensitivity and resistance for antibiotics

| Antibiotics | S, n (%) | R, n (%) | I/S, n (%) | Total, n (%) |
|-------------|----------|----------|------------|--------------|
| Amoxicillin | 16 (8.8) | 166 (91.2) | 0 | 182 (100) |
| Amoxicillin + clavulanic acid | 24 (13.2) | 158 (86.8) | 0 | 182 (100) |
| Methicillin | 31 (17)  | 151 (83)  | 0 | 182 (100) |
| Vancomycin  | 39 (21.4) | 143 (78.6) | 0 | 182 (100) |
| Meropenem   | 76 (41.8) | 95 (52.2)  | 12 (6.6) | 182 (100) |
| Imipenem    | 90 (49.5) | 79 (43.4)  | 17 (9.3) | 182 (100) |
| Ceftriaxone | 41 (22.5) | 141 (77.5) | 0 | 182 (100) |
| Ceftazidine | 16 (8.8)  | 166 (91.2) | 0 | 182 (100) |
| Cefepime    | 9 (4.9)   | 173 (95.1) | 0 | 182 (100) |
| Amikacin    | 53 (29.1) | 129 (70.9) | 0 | 182 (100) |
| Ciprofloxacin | 48 (26.4) | 134 (73.6) | 0 | 182 (100) |
| Piperacillin + tazobactam | 107 (58.8) | 73 (40.1)  | 0 | 182 (100) |

n=Total number, I/S=Intermediate sensitivity, S=Sensitivity, R=Resistance

S. aureus, E. coli, Acinetobacter, methicillin-susceptible S. aureus, and S. pneumoniae. S. aureus, and S. pneumoniae. Acinetobacter spp. were found to be the most common pathogen in VAP, followed by P. aeruginosa and S. aureus by Rit et al. [2]

Various factors affecting the pattern and organisms are host-microbial flora, prolonged antibiotic administration, and different ICU settings. Superinfection with MDR pathogens is commonly seen after prolonged antibiotic administration to ICU patients for primary infection. These findings are well corroborated with the findings of other studies which also noted that prior antimicrobial therapy markedly increased the rate of VAP caused by P. aeruginosa and Acinetobacter spp., and most of them are MDR pathogens. Goel et al. described susceptibility profiles of the etiological agents as colistin, followed by Piperacillin/ tazobactam and imipenem. They also mentioned that prior antibiotic therapy and prolonged hospitalization favor for
infection with MDR pathogens. Furthermore, the American Thoracic Society guidelines had mentioned that prior antibiotics or prior hospitalization within the past 90 days is at greater risk for colonization and infection with MDR pathogens.

The antibiotic sensitivity and resistance pattern is variable for different ICU as there are different criteria for diagnosis and management protocol. Furthermore, the patient subsets, referral cases, the ICU care, and many other factors are variable in different ICU. Hence, it is essential to generate local data.

In our study, *K. pneumoniae*, *Pseudomonas*, and *Acinetobacter* showed maximum sensitivity to piperacillin + tazobactam, followed by carbapenem and maximum resistance to cephalosporin, cefepime, and ceftriaxone. Goel et al. mentioned that colistin was found to be most effective antibiotic, followed by piperacillin/tazobactam combination and imipenem. Similar findings also noted in other recent studies. In our study, colistin was not studied, however, other susceptibility patterns are similar to other studies. The antibiotic sensitivity and resistance pattern differed for individual organism. The bacteriological approach for the management of VAP helps in deciding the appropriate antibiotics and may help in preventing resistance. In suspected nosocomial pneumonia, early culture and the study of antibiotic susceptibility pattern of the isolated organisms is essential.

Thus, the knowledge of these patterns helps in judicious selection and rational use of appropriate antibiotics. Gadani et al. mentioned that the de-escalation strategy (initiation of a broad-spectrum antibiotic and changing to a narrow spectrum after the sensitivity reports) will reduce inappropriate antibiotic use and subsequently the drug-resistant pathogens.

Thus, it is essential to generate local ICU data at regular intervals to choose empirical and appropriate antimicrobial therapy. A strict hospital infection control policy should be emphasized including judicious and rational antibiotic therapy, optimum preventive measures, and staff education programs.

The study was not without limitations as it was a single-center MICU study which did not include surgical and trauma patients. Antibiotic sensitivity and resistance pattern was studied for limited antibiotics. Study for other higher and newer antibiotics would have given better idea about the use of these antibiotics in MDR and difficult to treat cases. Furthermore, our study may not have the power to recognize all the risk factors associated with nosocomial pneumonia. Study done in different ICUs and inclusive of newer antibiotics testing would provide us the wider perspective.

**Conclusion**

Nosocomial pneumonia showed high incidence (17.44%) and mortality (60%). Common organisms identified were *S. aureus* and *K. pneumoniae*. Resistance was high for commonly used antibiotics and high antibiotic sensitivity for piperacillin + tazobactam and carbapenem. Thus, from the inference of the current study to reduce the incidence and mortality of nosocomial pneumonia, we recommend avoiding unnecessary prolonged hospitalization, intubation, and MV of the patient, whenever possible considering NIV and starting an early and appropriate weaning protocol. Furthermore, identifying risk factors for VAP and meticulous preventive measures in high-risk groups are must along with adequate staffing and strict hospital infection control policy. Knowledge of the prevalent organisms and sensitivity patterns in ICU helps in judicious and rational use of antibiotics which may reduce subsequent VAP.

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**Conflicts of interest**

There are no conflicts of interest.

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