**Microbial Dynamics and Antibiotic Resistance Patterns in Acute COPD Exacerbations; A prospective cohort study**

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**Competing interest statement**- All authors have completed the Unified Competing Interest form (available on request from the corresponding author). No financial relationships with any organizations that might have an interest in the submitted work in the previous three years and no other relationships or activities that could appear to have influenced the submitted work.

**Transparency declaration** - I, Alok Yadav, the lead author and guarantor of this manuscript, affirm that this is an honest, accurate, and transparent account of the study being reported. No important aspects of the study have been omitted, and any discrepancies from the study as originally planned (and, if relevant, registered) have been fully explained.

**Institutional Review Board statement:** Ethical clearance provided by Institutional Review Board of Manipal College of Medical Sciences.

**Informed consent statement:** Informed written consent was obtained from all participants and study received ethical clearance from the institutional review board.

**Ethical Consideration Statement**: All the participants provided informed written consent prior to their enrollment in the study, which included clear information about potential of publication of anonymized data. Participants were informed that no identifiable personal information will be disclosed in any publications of the study. Given that many participants reside in distant and rural areas and may not return for follow-up, it was not feasible to obtain re-consent specifically for manuscript publication. However, we have ensured full anonymization of data, and no individual patient is identifiable in any form.

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**Patient Involvement**: Patients were not involved in the initial design, recruitment and conduct of the study. The study questions were shaped by clinical observations of frequent antibiotic resistance and poor empirical therapy response in patients hospitalized with AECOPD in Manipal Teaching Hospital. Recurrent concerns regarding the failure of empirical antibiotics, delays in microbiological diagnosis, burden of repeated hospital admissions and prolonged hospital stay highlighted the need to explore microbiological etiology and resistance pattern in our setting. During the study, participants were provided with clear information regarding the purpose and potential impact of the research. All the participants provided informed written consent. Although patients were directly not involved in data analysis and interpretation, their clinical influences strongly influenced the research direction. We intend to disseminate the findings of the study to the participants and the public via information leaflets, community outreach programs in simple Nepali languages.

**Data Avaibility:** The data generated and analyzed during the study are available from the corresponding author on reasonable request. There are no ethical, privacy and security concerns preventing the release of these data.

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**Abstract**

Introduction

AECOPD is associated with an increased rate of hospitalization, readmission, rapid decline in lung function, and worsened quality of life. Bacterial infection is a key trigger, making it important to evaluate its incidence and resistance patterns across centers.

Methodology

A hospital-based cross-sectional study was conducted among AECOPD patients at Manipal Teaching Hospital, Nepal, after IRC approval and informed written consent. Sputum and blood samples were collected for microbiological and inflammatory marker analysis, with data analyzed using SPSSv 25.

Results

A total of, 396 cases aged 48 to 97 years were analyzed, with higher incidence in females (53.9%) than males (46.1%). The mean age of exacerbation was significantly higher in males (75.33 years) than females (73.11years) with p=0.012(<0.05).  Bacterial growth was observed in 31 % (n=123). Maximum (87.5%) bacterial growth was observed with purulent sputum, and chi square (χ2) test between sputum consistency and culture growth showed p<0.05 and phi value (0.689) respectively, indicating strong association. Common isolates included *Acinetobacter baumani complex* (26.83%), *Klebsiella pneumoniae* (21.95%), *Pseudomonas aeruginose* (20.33%), *Staphylococcus aureus* (15.45%). Among the isolated organisms, 42.27% were MDR, and 20.32% were XDR. 94.7% of the isolated Staphylococcus aureus were MRSA with MDR.

Conclusion

This study highlights the predominance of gram-negative bacteria and alarming rates of MDR/XDR strains, including high CA-MRSA, in AECOPD. Purulent sputum, increased WBC counts were strong positive predictors of bacterial etiology. The high resistance patterns underscore the need for institution-specific antibiograms, robust antibiotic stewardship and continued local surveillance to guide effective empirical therapy and combat rising antimicrobial resistance.

Abbreviations

AECOPD**:** Acute exacerbation of COPD

CA-MRSA: Community Acquired Methicillin Resistant Staphylococcus aureus

MDR: Multi drug resistant

XDR: Extensively drug resistant

keywords

Microbial

AECOPD

Antibiogram

Bacterial

Antibiotic sensitivity pattern

**Introduction**

Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production, and/or exacerbations) due to abnormalities in the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction. (1)  
The overall prevalence of COPD is 10.3 % worldwide (2).COPD is one of the top three causes of mortality per annum, with three million deaths per annum. Mortality from COPD and related conditions is expected to increase to 5.4 million by 2060(1). Nepal has the highest age-standardized death rate and the highest age-standardized disability-adjusted life-year rate, which is three times higher than the global DALY rate (1, 3).  
Exacerbation of chronic obstructive pulmonary disease (ECOPD) is defined as an event characterized by increased dyspnea and/or cough and sputum that worsens in < 14 days, which may be accompanied by tachypnea and/or tachycardia and is often associated with increased local and systemic inflammation caused by infection, pollution, or other insults to the airways (1,4). Exacerbation of COPD has been associated with an increased rate of hospitalization, readmission, rapid decline in lung function, worsened quality of life, and decreased exercise performance (5). Furthermore, the strongest predictor of the risk of future exacerbation was a history of exacerbation itself (6). Indeed, a longer duration of exacerbation is associated with poorer health status and an increased risk of new events. (7) Thus, proper management of exacerbations is essential for prognosis.  
Although, infectious agents (bacteria, viruses, and fungi) are the predominant causes of exacerbation, smoking, (8) environmental factors such as air pollution and excessive heat, are also implicated as risk factors (1,9). The Bacterial infection is estimated to cause 49.59% of exacerbations of COPD (10).

Furthermore, purulence of sputum, increased CRP levels were associated with higher rates of isolation of bacteria in sputum culture (11). Commonly isolated bacteria from sputum samples in acute exacerbation of COPD include *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Acinetobacter species*, *Streptococcus pneumoniae*, and *Escherichia coli*. Various studies have shown an increasing proportion of resistance to multiple antibiotics among the species of bacteria isolated from sputum samples of AECOPD. (12, 13) The variability in microbial profiles and antibiotic resistance patterns between geographical regions and populations constitutes critical knowledge gap, which may help to provide adequate antibiotic therapy.

This study aimed to analyze the burden of antimicrobial resistance in COPD, where the recurrent use of antibiotics is required, and to question the current policy of empirical antibiotic therapy during exacerbations. This study aims to isolate and identify bacterial agents associated with AECOPD, analyze factors pointing towards the bacterial etiology of AECOPD, determine antibiotics resistance patterns of bacterial isolates and establish a proper framework for use of antibiotics in the management of AECOPD and to report any demographic findings relevant to AECOPD cases.

**Methodology**

An observational study was conducted among patients admitted to the pulmonology unit of the Medicine Department at the Manipal Teaching Hospital, Nepal from October 2023 to May 2024 after receiving approval from the Institutional Review Committee of Manipal college of medical sciences, Pokhara. The IRC provided approval under the ethical board approval number IRC/MCOMS/578. Patients were provided with comprehensive information regarding the study objectives and procedures, and informed written consent was obtained to ensure voluntary participation.  
Employing a hospital-based prospective cohort study design, the research focused on a specific subset of patients admitted with Acute Exacerbation of Chronic Obstructive Pulmonary Disease (AECOPD). Strict inclusion criteria were applied to ensure the accuracy and reliability of the data. Patients who had not received antibiotics in the preceding month were included in the study cohort. Patients with increased dyspnea and/or cough and sputum that worsen in < 14 days, which may be accompanied by tachypnea and/or tachycardia and is often associated with increased local and systemic inflammation caused by infection, pollution, or other insults to the airways. To gather comprehensive insights into the patients' clinical status, questionnaires were used to document patient’s demographics, detailed history for their symptoms and past antibiotic usage.

Sputum samples were collected from participants after deep coughing using sterile, transparent, multipurpose specimen containers. The patients were asked to rinse their mouths properly before sputum collection. The gross appearance of each sputum sample, that is, color and consistency, was noted to provide a valuable context for subsequent microbiological testing. These samples were promptly dispatched to the microbiology laboratory for culture and antibiotic susceptibility testing, using the disc diffusion method.  
As per the antibiograms, MDR and XDR were evaluated as MDR was defined as acquired non susceptibility to at least one agent in three or more anti-microbial categories, and XDR was defined as non-susceptibility to at least one agent in all but two or fewer antimicrobial categories.  
Additionally, to recognize the potential correlation between exacerbations and systemic inflammation, blood samples were collected for total leukocyte count. This allowed for a comprehensive exploration of the relationship between the total leukocyte count and exacerbations attributed to bacterial etiology. Upon completion of the data collection, data entry and analysis were conducted using SPSS version 25. The statistical analysis includes frequency analysis of demographic data, chi square test for analysis of categorical (nominal) data, p value and phi value were used to signify association between two variables and strength of association. Similarly-test was used to compare means of two independent groups.

Sample size calculation:

Sample size (n)

n= z2pq/e2

z=statistic for level of confidence

p=prevalence of acute exacerbation of COPD

e= absolute allowable error

q=100-prevalence (if in percentage)

= (1-prevalence if in decimal)

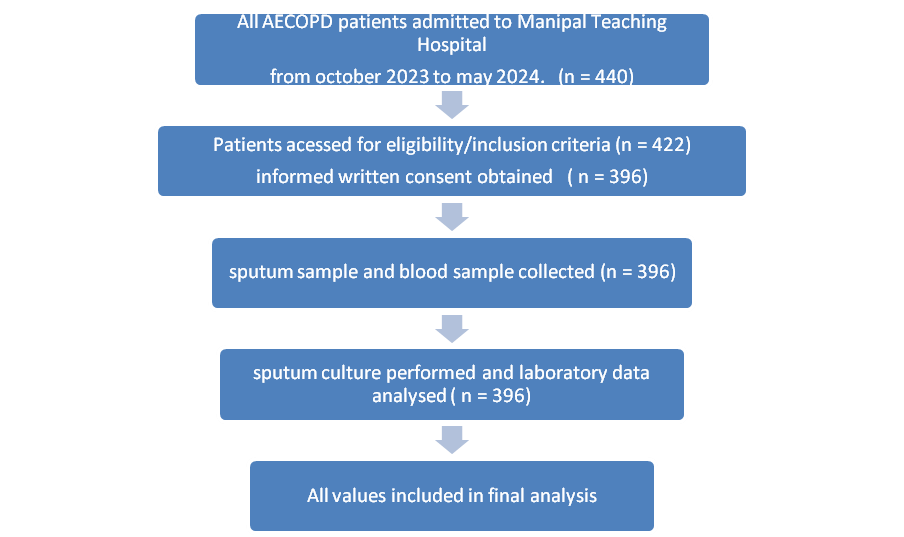
Prevalence =50% (14)

q= (100-50) =50%

Confidence interval taken=95%

Error=5%

z=1.96

n= (1.96)2\*50\*50/5\*5 =384.16=384 cases

1: Flow diagram of cases selection and cases exclusion

Out of 440 patients admitted from October 2023 to May 2024 in pulmonology unit with AECOPD, 18 people had well documented recent use of antibiotic and did not match the eligibility criteria. Out of remaining 422 cases, 26 patient/patient’s party were unwilling to give consent. Thus total cases remaining after obtaining written consent were 396 who were included in the study as shown in figure 1.

**Results**

In this study, 396 patients were aged 48 - 97 years with a mean age of 74.14 years were analyzed. The largest group (41.1%) fell within the age group of 71-80 years, with 46.1% male and 53.9% female. An independent sample t-test showed that the mean age of exacerbation for males (75.33 years) was higher than that for females (73.11years) with p=0.012(<0.05).  
  
Bacterial growth was observed in 31.81% (126) cases. Notably, white sputum showed no growth in 87.1% cases, whereas yellow, green, and blood-tinged sputum showed 71%, 80%, and 0%growth, respectively. The chi-square (χ2) test between sputum color and culture reports showed a p value<0.05, with a phi value(0.633) indicating a strong association between sputum color and culture report.  
  
Sputum consistency also correlated with culture results, with purulent sputum having highest 87.5% followed by mucopurulent, mucoid, and serous at 57.1%, 23.1% and 0.7% respectively, with the chi-square test showing (χ2) p<0.05, and phi=0.689.  
Raised leukocyte count was associated with positive bacterial growth, as bacterial growth was observed in 20.3%, 31.8%, 47.9% of cases showed positive bacterial culture when the total leukocyte count was between 4,000to 11,000/[cu mm](http://cu.mm/),<4,000/[cu mm](http://cu.mm/) and >11,000/cumm respectively. The Chi-square (χ2) test between the total leukocyte count and culture results showed a p-value of <0.05.  
The most commonly isolated organisms were *Acinetobacter baumani complex*(26.19%), *Klebsiella pneumoniae*(21.42%), *Pseudomonas aeruginosa* (19.84*%), Staphylococcus aureus* (15.07%), *Escherechia coli* (12.69%), and others like *Streptococcus pneumonia and Citrobacterium freundii*is depicted in Figure 2.

Figure 2: Pie chart showing frequency of bacterial isolates.

Antibiotic sensitivity test reports revealed that *Acinetobacter baumani complex* showed the highest sensitivity to colistin (93.9%), polymyxin B(93.9%), amikacin (48.5%), followed by carbapenams (36.4%), and piperacillin-tazobactam (39.4%), with 30.3% multi drug resistant (MDR) and 42.4% extensively drug resistant (XDR) as shown in Table 1.  
*Klebsiella pneumonia showed* the highest sensitivity to amikacin (96.2%), followed by carbapenams (92.6%), and piperacillin-tazobactam (92%), with 24.24% MDR strain and 39.39% XDR strain as illustrated in table 2.

*Pseudomonas aeruginosa* showed the highest sensitivity to amikacin (92%), carbapenams (92%), polymixin B and colistin (92%) and piperacillin-tazobactam (80%), with 24.0% MDR and 4% XDR strain.  
*Escherichia coli* also showed the highest sensitivity to tigecycline(100%), colistin(100%), polymixin B(100%) followed by amikacin (75%), carbapenams (75%), and piperacillin-tazobactam (58.3%), with 62.5% MDR strain and 12.5% XDR strain whereas only 25% of the strain were non-MDR non-XDR.  
*Staphylococcus aureus* showed sensitivity to linezolid(100%), teicoplanin(100%), vancomycin(94.73%), amikacin (72.2%). The sputum sample was collected soon after hospital admission within 24 hours, thus the isolated species were community acquired. 94.73% of the *S. aureus* species were MRSA with 57.89% MDR strain and 36.84 % XDR strain. Only 5.2% of the isolated strain of *S. aureus* were non-MDR strain as shown in Table 1 and Table 2.

42.27% and 20.32% of total species isolated were MDR and XDR strain respectively.  
*Candida albicans* was isolated from 21 specimens (5.30%), with a mean age of 77.14 years, and male-to-female ratio of 2:1. In cases where *Candida albicans*  was isolated, 71.4% had whitish sputum, and 42% had mucoid consistency, and 57% of cases, a WBC count was >11,000/cumm.

**Discussion:**  
We observed higher incidence of COPD exacerbations among females(53.9% ) compared to males (46.1%) in contrast with most of previous studies, which typically reported higher rates among males.(14,15) This difference could be multi-factorial. Some studies attributes rising incidence among females is cumulative effect of increased rates of smoking and more deleterious effect of smoking on lung health in female. (16, 17) This is also implicated in our study where male has significantly higher mean age of exacerbation in comparison to female (p value < 0.05) indicating difference in progression of diseases in male and female which must be further explored in different study. In context of Nepal, use of firewood and biomass fuel use can also be significant cause of this trend. (18)

45.32% of the female admitted with AECOPD use firewood and biomass fuel for cooking. However, smoking habits of female and use of firewood for cooking were not systemically assessed in our study, as our primary objective was bacteriological profile and their antibiotic sensitivity of AECOPD cases. Thus, further studies should evaluate the effect of epidemiological and risk factors among male and female especially with high biomass fuel exposure.  
  
The analysis of sputum samples revealed bacterial growth in 31% cases, similar to some studies showing 30%(1), but different from others with 50-60%. (14,19) The study revealed distinct patterns in bacterial growth corresponding to different sputum consistencies, where bacterial isolation was maximum in purulent and least in serous sputum, with a chi-square test showing p<0.05, and phi= 0.698, indicating a strong association. These findings are consistent with those of the previous studies. (20,21) Our findings align with the Anthonisen criteria for sputum purulence, which has long been a reliable predictor of bacterial infection in COPD exacerbation, guiding treatment decisions. (22) Chi-square test showed a significant association with increased leukocyte count (>11,000/cumm) and bacterial growth as well (p<0.05). Thus, sputum purulence and leukocyte count can be used as guiding tool for antibiotic use as only 30% to 50% of the cases have bacterial infective etiology.

The main bacterial isolates were *Acinetobacter baumani* complex (26.83%), *Klebsiella pneumonia* (21.95%), *Pseudomonas aeruginosa* (20.33%), *Staphylococcus aureus* (15.45%), and *E. coli* (9.76%), with gram-negative bacteria accounting for 82.11% of the isolates. The predominance of *Acinetobacter baumani* complex is consistent with other studies conducted especially those conducted in Nepal. (23,24). In contrast*, Klebsiella* and *Pseudomonas* species have been reported as most common pathogen in several other studies done at different healthcare institutions at different geographical locations. (12,13) These variations suggest that the bacteriological profile of AECOPD differs not only from study to study but also between healthcare institutions, although some patterns tend to reflect geographical similarities.

Thus, different institutions must develop and maintain their own antibiograms, which could be helpful to guide appropriate antibiotic therapy for common isolates for AECOPD cases at their respective institutions and comparison should be done with other institutes for common geographical distribution of isolates. Furthermore, regular inter-institutional comparisons, particularly within same geographical region could facilitate better understanding of regional microbial patterns.

A high burden of antibiotics resistance was observed among *Acinetobacter baumani* complex, consistent with findings from other studies. (23, 25) The sensitivity patterns of *Pseudomonas aeruginosa* and *Escherichia coli* as in Table 1 had different sensitivity patterns from the published literature. (14, 15, 26) Similarly, the antibiotic sensitivity patterns of *Klebsiella pneumonia* as in Table 2 showed slight variation from those reported in the existing literature. (19,26)  
These discrepancies underscore the dynamic nature of antimicrobial resistance, which can vary significantly. Thus, this discrepancy highlight the significance of targeted antibiotic therapy guided by local/institutional resistance patterns for better management of AECOPD. Such measures will support clinicians in selecting appropriate antibiotics, limiting use of broad spectrum empirical antibiotics, which may help to halt the rising tide of antimicrobial resistance.

As 94.73% of the *Staphylococcus aureus* isolates were MRSA and since the sputum samples were collected at the time of hospital admission, these MRSA strains are likely to be community acquired. The isolation of such high proportion of CA-MRSA (94.73%) among AECOPD cases raises serious concern regarding current guidelines for antibiotic use in AECOPD, especially considering that bacterial etiology accounted for only 30% of cases in this study. Various study has shown that the isolation of MRSA was associated with worse outcome among patient of AECOPD. (27) More than half (62.6%) of the total isolated strains were either MDR or XDR i.e. 42.27% and 20.32% respectively.

This increasing trend of antibiotic resistance has important implications for both clinical practice and public health. (28) Routine empirical use of broad spectrum antibiotics in AECOPD may drive further antimicrobial resistance. Thus, to avoid such high burden of resistance cautious use of antibiotics among AECOPD cases are required. Limiting antibiotic use to patients with clear evidence of bacterial infection i.e. culture positive cases or antibiotic use restricted to high risk cases such as cases with purulent sputum or increased leukocyte count could help reduce the burden of resistance organisms, especially MRSA (94.7% of staphylococcus aureus strains).  
The detection of *Candida albicans* as a potential etiological factors in AECOPD add complexity to interpretation of our findings. (29) Our findings indicate a notable correlation between presence of *Candida* and elevated WBC count (p value < 0.05) highlighting a potential inflammatory response. A similar association has been reported by other investigators and claims potential role of use of steroids. (1,29,30) These findings underscore the need for further investigation into clinical significance of *Candida* colonization or infection in AECOPD and its potential impact on disease management.

Limitations of the Study  
1.This study focused only on bacteria and Candida and did not account for viral and other fungal infections.  
2. Over-the-counter antibiotic use was not documented, potentially leading to culture-negative reports.  
3. The use of antibiotics during previous exacerbations was not taken into account, which may have influenced the results.

Proposal for new research

1. Investigate development and progression of COPD among males and females.
2. Explore the role of Candida as pathogen or normal flora in AECOPD and its relation to steroid use.
3. Comparative analysis of (a) empirical treatment in all cases of AECOPD vs. (b) evidence-based therapy vs. (c) empirical treatment in the high-risk group as per the clinical presentation.
4. Develop and follow guidelines for minimizing the use of antibiotics and reducing antibiotic resistance.

**Conclusion**

Purulent sputum, increased WBC counts were strong positive predictors of bacterial etiology. Gram-negative bacteria were the most predominant isolates (*Acinetobacter baumani*, followed by *Klebsiella pneumoniae*) and the rising incidence of CA-MRSA, MDR and XDR strains calls for a reevaluation of current antibiotic policies. The higher incidence of AECOPD among females and earlier mean age of presentation in female compared to males suggests need for further evaluation of COPD progression in different sexes. The role of Candida in AECOPD should be further evaluated. The variation in common bacterial pathogen and their antibiotic sensitive patterns signifies the importance of regular local microbial surveillance and inter-institutional data sharing.

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|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | *Acinetobacter baumani complex* | | *Pseudomonas aeruginosa* | | *Staphylococcus aureus* | | *Escherechia Coli* | |
|  |  | sensitive | Resistance | sensitive | resistance | sensitive | resistance | Sensitive | Resistance |
| Aminoglycoside | Amikacin | 16(48.5%) | 17 (51.5%) | 23(92%) | 2(8%) | 14 | 5 | 12(75%) | 4(25%) |
| Gentamycin | 16(48.5%) | 17(51.5%) | 23(92%) | 2(8%) | 14 | 5 | 12(75%) | 4(25%) |
| Tobramycin | 16(48.5%) | 17(51.5%) | 23(92%) | 2(8%) | 14 | 5 | 12(75%) | 4 |
| Fluorouinolones | Ciprofloxacin | 10(30.3%) | 23(69.7%) | 16(64%) | 9(36%) | 0 | 19 | 6(37.5%) | 10(62.5% |
| Levofloxacin | 17(51.5%) | 16(48.5%) | 20(80%) | 5(20%) | 9 | 10 | 7(43.75%) | 9(56.25%) |
| Beta lactams | Penicillins | 5(15.15%) | 28(84.85%) | 4(16%) | 21(84%) | 2 | 17 | 4(25%) | 12(75%) |
| Cephalosporin | 8(24.24%) | 25(75.76%) | 15(60%) | 10(40%) | 1 | 18 | 5(31.25%) | 11(68.75%) |
| Carbapenems | 12(36.4%) | 21(63.6%) | 23(92%) | 2(8%) | 6 | 14 | 12(75%) | 4(25%) |
| Monobactams | 13(39.4%) | 20(60.6%) | 21(84%) | 4(16%) | - | - | 11(68.75%) | 5(31.25%) |
| Glycopeptides | Vancomycin | - | - | - | - | 18 | 1 | - | - |
| Teicoplanin | - | - | - | - | 19 | 0 | - | - |
| Macrolides | Azithromycin/ Erythromycin | 12(36.4%) | 21(63.6%) | 3(12%) | 22(88%) | 17 | 2 | 5(31.25%) | 11(68.75%) |
| Tetracyclines | Tigecycline | 28(84.8%) | 5(15.2%) | 23(92%) | 2(8%) | 19(100%) | 0 | 16(100%) | 0 |
| Polymixins | Colistin | 31(93.9%) | 2(6.1%) | 23(92%) | 2(8%) | - | - | 16(100%) | 0 |
| Polymyxin B | 31(93.9%) | 2(6.1%) | 23(92%) | 2(8%) | - | - | 16(100%) | 0 |
| Others | Sulfonamide | 7(21.2%) | 26(68.8%) | 15(60%) | 10(40%) | 9 | 10 | 10(100%) | 6 |
| Linezolid | - | - | - | - | 19 | 0 | - | - |
| Non- MDR |  | 11(33.33%) | | 18(72%) | | 1(5.2%) | | 4(25%) | |
| MDR |  | 8(24.24%) | | 6(24%) | | 11(57.89%) | | 10(62.5%) | |
| XDR |  | 13(39.39%) | | 1(4%) | | 7(36.84%) | | 2(12.5%) | |
| PDR |  | 1(0.03%) | | 0 | | 0 | | 0 | |

Table 1: Antibiotic sensitivity for *Acinetobacter baumani complex, Pseudomonas aeuroginosa, Staphylococcus aureus, Escherechia coli*

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | *Klebsiella pneumonia* | | *Streptococcus pneumonia* | | *Citrobacter* | |
|  |  | sensitive | Resistance | Sensitive | Resistance | Sensitive | resistance |
| Aminoglycoside | Amikacin | 26(96.29%) | 1(3.71%) | 2(66.67%) | 1(33.33%) | 3(100%) | 0 |
| Gentamycin | 26(96.29%) | 1(3.71%) | 2(66.67%) | 1(33.33%) | 3(100%) | 0 |
| Tobramycin | 26(26.29%) | 1(3.71%) | 2(66.67%) | 1(33.33%) | 3(100%) | 0 |
| Fluorouinolones | Ciprofloxacin | 15(55.56%) | 12(44.44%) | 1(33.33%) | 2(66.67%) | 3(100%) | 0 |
| Levofloxacin | 18(66.67%) | 9(33.33%) | 3(100%) | 0 | 3(100%) | 0 |
| Beta lactams | Penicillins | 11(40.74%) | 16(59.26%) | 1(33.33%) | 2(66.67%) | 1(33.33%) | 2(66.67%) |
| cephalosporin | 23(85.18%) | 4(14.82%) | 2(66.67%) | 1(33.33%) | 2(66.67%) | 1(33.33%) |
| Carbapenems | 25(92.59%) | 2(7.41%) | 3(100%) | 0 | 3(100%) | 0 |
| Monobactams | 24(88.89%) | 3(11.11%) | - | - | - | - |
| Glycopeptides | Vancomycin | - | - | 3(100%) | 3(100%) | - | - |
| Teicoplanin | - | - | 3(100%) | 3(100%) | - | - |
| Macrolides | Azithromycin/ Erythromycin | 10(37.04%) | 17(62.96%) | 1(33.33%) | 2(66.67%) | 2(66.67%) | 1(33.33%) |
| Tetracyclines | Tigecycline | 27(100%) | 0 | 2(66.67%) | 1(33.33%) | 3(100%) | 0 |
| Polymixins | Colistin | 27(100%) | 0 | - | - | 3(100%) | 0 |
| Polymyxin B | 27(100%) | 0 | - | - | 3(100%) | 0 |
| Others | Sulfonamide | 16(59.25%) | 11(40.75%) | 1(33.33%) | 2(66.67%) | 3(100%) | 0 |
| Linezolid | - | - | 3(100%) | 0 | - | - |
| Non-MDR |  | 12(44.44%) | | 0 | | 2(66.67%) | |
| MDR |  | 13(48.15%) | | 3(100%) | | 1(33.33%) | |
| XDR |  | 2(7.4%) | | 0 | | 0 | |
| PDR |  | 0 | | 0 | | 0 | |

Table 2: *Antibiotic sensitivity for Klebsiella pneumoniae, Streptococcus pneumoniae, Citrobacter sps*