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BACTERIOLOGICAL PROFILE AND ANTIBIOGRAM OF VENTILATOR ASSOCIATED PNEUMONIA IN INTENSIVE CARE UNIT PATIENTS.

Microbiology

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ABSTRACT

Background: Ventilator associated pneumonia (VAP) is the most common ICU acquired pneumonia that is reported to occur in nearly 9-27% of patients on mechanical ventilator. Aetiology of VAP varies greatly as per geographical area, duration of mechanical ventilation, and onset (early/late) of VAP.6 As compared to community acquired infections, bacteria causing HAI are significantly multi drug resistant (MDR), making the treatment more complicated. The present study attempts to determine the bacteriological profile and its antimicrobial susceptibility pattern of VAP. **Methods:** The study included a total of 100 adult patients (≥ 18 years) with mechanical ventilator for >48 hrs. Endotracheal aspirate (ETA) obtained from these patients were obtained and subjected to Gram staining and culture. The antimicrobial susceptibility profile of the isolates was performed by Kirby-Bauer disc diffusion method on Mueller Hinton agar. **Results:** VAP was diagnosed 29% of patients on the basis clinical pulmonary infection score (CPIS) confirmed cases. The most frequently isolated organisms in VAP patients were Acinetobacter baumannii followed by Pseudomonas aeruginosa, Klebsiella pneumoniae and E. coli. In early onset VAP 5(50%) pathogens were MDR and in late onset VAP 13(68.42%) pathogens were MDR. **Conclusion:** Mechanically ventilated patients are at high risk of developing ventilator associated pneumonia (VAP), as 29% of patients developed VAP in this study. Late onset VAP is more common than early onset VAP. Acinetobacter baumannii is the commonest isolate from VAP cases. There is great variation antimicrobial susceptibility pattern of isolates from late onset VAP and early onset VAP. Hence it is necessary to understand the bacteriological profile and their susceptibility for designing the guidelines for prophylaxis and treatment of VAP cases.

KEYWORDS

Ventilator Associated Pneumonia, Healthcare Associated Infection, Antimicrobial Susceptibility Profile, Bacteriological Profile.

INTRODUCTION

Healthcare associated infections (HAI) are the most common to threat to patient safety. These infections detrimentally add to morbidity, mortality, healthcare cost and length of hospital stay. For the purpose of surveillance in a healthcare setting, the Center for Disease Prevention and Control (CDC) National Health Surveillance Network (NHSN) defines HAI as 'a localized or systemic condition resulting from an adverse reaction to the presence of an infectious agent(s) or its toxin(s). There must be no evidence that the infection was present or incubating at the time of admission to the acute care setting'.[1]

HAI can be caused by variety of pathogens that may be either endogenous or exogenous in origin. Catheter associated urinary tract infection (CAUTI), central line associated blood stream infection (CLABSI), ventilator associated pneumonia (VAP) and surgical site infection (SSI) is common HAI. Among various types of HAI, VAP is associated with high mortality rates (24-76%).[2]

VAP is the most common ICU acquired pneumonia that is reported to occur in nearly 9-27% of patients on mechanical ventilator.[3] Aetiology of VAP include Gram negative bacteria like Acinetobacter baumannii, Pseudomonas aeruginosa, Klebsiella pneumonia and Escherichia coli, and Gram positive cocci like Staphylococcus aureus.[5] Researchers have reported that aetiology of VAP varies greatly as per geographical area, duration of mechanical ventilation, and onset (early/late) of VAP.[6] As compared to community acquired infections, bacteria causing HAI are significantly multi drug resistant (MDR), making the treatment more complicated.[7] Therefore it is highly essential to know the pattern of pathogens associated with VAP along with antimicrobial susceptibility profile for rationalising the antimicrobial therapy for improved clinical outcome of the patient. The present study attempts to determine the bacteriological profile and its antimicrobial susceptibility pattern of VAP.

MATERIAL AND METHODS

This prospective study with descriptive cross-sectional study design was conducted in the Department of Microbiology in collaboration with medical and surgical ICU s for a period of 21 months (October 2019 – June 2021). The protocol of the study was approved by Institutional Ethics Committee. The study included a total of 100 adult patients (≥ 18 years) with mechanical ventilator for >48 hrs. The inclusion and exclusion criteria of the study were as follows.

Inclusion Criteria

- Adult patients (≥ 18 years) of both sexes.
- On mechanical ventilation for >48 hours
- Radiological, haematological and clinical parameters indicative of VAP
- Tracheostomised patients with suspected VAP.
- Extubated patients with suspected VAP.

Exclusion Criteria

- Patients < 18 years of age
- Known case of pulmonary tuberculosis.
- Patients with pneumonia prior to mechanical ventilation or <48 hours of mechanical ventilation.
- Patients with other causes of radiological infiltrates like pulmonary embolism, pulmonary haemorrhage, atelectasis, congestive heart failure and adult respiratory distress syndrome
- Patients with severe immunocompromised conditions like AIDS, organ transplant, terminal stages of malignancy.

Endotracheal aspirate (ETA) obtained from these patients were aseptically collected from these patients and sent to laboratory. These ETA samples were subjected to Gram staining and culture.

For isolation, identification and categorization of pathogens, the ETA samples were first diluted with sterile normal saline 1/10, 1/100, 1/1,000 times. A concentration of 0.01 ml of these dilutions was then aseptically inoculated onto the culture media like 5% sheep blood agar, nutrient agar, Macconkey agar and chocolate agar.

Upon inoculation, the culture plates were incubated at 37°C for 24-48 hours. After incubation the colony count was made number of colony forming units per ml (CFU/ml) was estimated. Growth of any organism $\geq 10^5$ CFU/ ml was considered as significant whereas growth $\leq 10^5$ CFU/ ml was considered as colonization or contamination.[8,9] The organism was identified as per standard microbiological protocol including colony characteristics, Gram nature, motility and biochemical reactions.

Gram-stained smears of ETA were observed for the presence of squamous cells, polymorph nuclear cells, and organisms. Upon Gram staining, ETA samples showing >10 PMNs / high power field, ≥ 1 bacterium / oil immersion field, and presence of intracellular bacterial inclusions were considered as indicative of VAP whereas samples showing squamous epithelial cells >10/ low power field and no organism / oil immersion field.

The antimicrobial susceptibility profile of the isolates was performed by Kirby-Bauer disc diffusion method on Mueller Hinton agar and

interpreted as per the Clinical and laboratory Standards Institute (CLSI) 2020 guidelines.

Patient's demographic characteristics (age, sex), relevant clinical features (date of admission, date of intubation/tracheostomy, clinical diagnosis, underlying diseases, duration of mechanical ventilation, prior antibiotic therapy, etc.), medical history (chief complaint, past history, general examination, systemic examination), assessment of level of consciousness, risk factors (e.g. presence of nasogastric tube, enteral nutrition, antacid or histamine type 2 i.e. H2 blocker therapy) and radiological and pathological investigations (Chest x-ray, CT scan, WBC count) were recorded and analysed. The clinical condition of the patients was followed up from the time of inclusion in the study to the date of discharge from hospital or death.

The data obtained from the study was entered in the Microsoft ® Excel spreadsheet program. Statistical analysis was performed by SPSS version 20.0, IBM, USA.

RESULTS

A total of 10 patients were grouped into 5 groups as per their age. 28% of patients included in the study belonged to age group >60 years (Figure 1). Male patients (65%) predominated over females (35%)

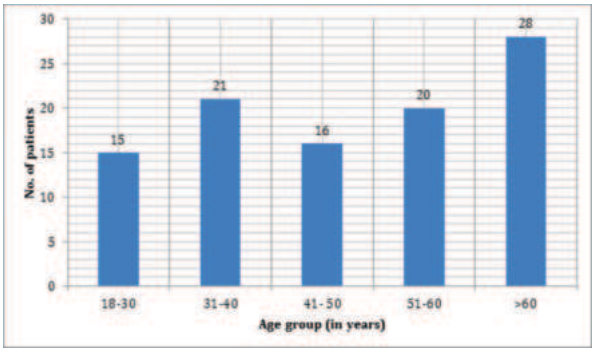

Figure 1. Age Wise Distribution of Patients.

The clinical spectrum of the patients was wide and included cases of neurological disorders (20%), poisoning (18%), sepsis (16%), central nervous system infections (13%), intra-abdominal diseases, (13%), cardiovascular diseases (10%), and head injury (8%).

Radiological diagnosis showed that 42% of patients had diffuse chest infiltration whereas 26% had localised chest infiltration. There was no evidence of chest infiltration in 32% of patients. A total of 89% of patients were intubated whereas tracheostomy was performed in 11% of patients. In 29 % the clinical pulmonary infection score (CPIS) was >6 and hence these patients were diagnosed as confirmed cases of VAP.

Confirmed VAP cases were common in age group 18-30 years (8%) age, followed by 41-50 years (7%) and 51-60 years (5%). Age group 31-40 years had 4 cases whereas remaining 5 belonged to age group >60 years. Out of 29 confirmed VAP cases, 19 (65%) were females whereas 10 (35%) were males.

A total of 19 (65.5%) patients had late onset VAP whereas 10 (34.5%) had early onset VAP. A total of 9 (31.1%) patients succumbed to the condition, 12 (41.4%) were treated and discharged from the facility whereas 8 (27.6%) opted for discharge against medical advice (DAMA). Presence of nasogastric tube, impaired consciousness and reintubation were common risk factors noted in confirmed cases of VAP (Figure 2).

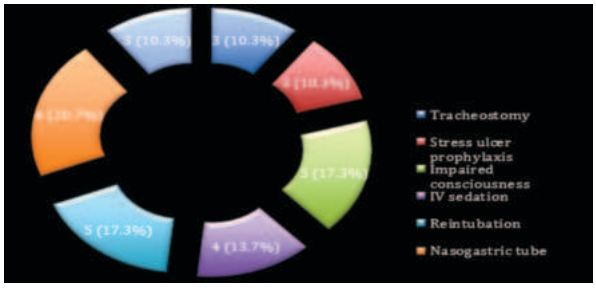

Figure 2. Risk Factors in Confirmed VAP Cases

As shown in table 1, Nil fermenters like Acinetobacter baumannii and Pseudomonas aeruginosa followed by Enterobacterals like Klebsiella pneumoniae and E. coli were common bacterial pathogens isolated from VAP cases. In the present Citrobacter freundii was not isolated from cases with late onset VAP.

Table 2. Bacteriological Profile of VAP.

Organisms	Total (%)
Acinetobacter baumannii	7 (24.1)
Pseudomonas aeruginosa	5 (17.2)
Klebsiella pneumoniae	5 (17.2)
E. Coli	4 (13.8)
Staphylococcus aureus	4 (13.8)
Coagulase negative Staphylococci (CoNS)	2 (6.9%)
Citrobacter freundii	2 (6.9)
Total	29

The bacteriological profile and antimicrobial susceptibility profile of early onset VAP is summarized in table 3. Citrobacter freundii was isolated only from cases of early onset VAP. All bacterial pathogens isolated from early onset VAP were susceptible to ceftazidime.

Table 3. Bacteriological Profile and Antimicrobial Susceptibility Profile of Early Onset VAP.

Antimicrobial agent	Acinetobacter baumannii (n=3)	Pseudomonas aeruginosa (n=2)	Klebsiella pneumoniae (n=1)	Citrobacter freundii (n=2)	E. coli (n=1)	S. aureus (n=1)
Amp	0(0%)	0(0%)	0(0%)	1(50%)	1(100%)	-
PIT	2(66%)	1(50%)	1(100%)	1(50%)	0(0%)	-
AK	2(66%)	1(50%)	1(100%)	1(50%)	0(0%)	-
CIP	2(66%)	2(100%)	1(100%)	1(50%)	1(100%)	1(100%)
CTX	2(66%)	1(50%)	1(100%)	1(50%)	0(0%)	-
CAZ	3(100%)	2(100%)	1(100%)	2(100%)	0(0%)	-
MRP	1(33%)	1(50%)	0(0%)	0(0%)	0(0%)	-
PB	3 (100%)	2 (100%)	1 (100%)	2 (100%)	1(100%)	-
TGC	1(33%)	1(50%)	1(100%)	1(50%)	1(100%)	0(0%)
GEN	2(66%)	1(50%)	1(100%)	1(50%)	0(0%)	-
TOB	2(66%)	1(50%)	1(100%)	1(50%)	0(0%)	-
COT	-	-	-	-	-	0(0%)
VA	-	-	-	-	-	0(0%)
LZ	-	-	-	-	-	0(0%)
AZ	-	-	-	-	-	0(0%)
CX	-	-	-	-	-	0(0%)
TEI	-	-	-	-	-	0(0%)
E	-	-	-	-	-	1(100%)
CD	-	-	-	-	-	1(100%)
P	-	-	-	-	-	1(100%)
TE	-	-	-	-	-	0(0%)

Amp = Ampicillin, P = Penicillin, PIT = Piperacillin-Tazobactam, AK = Amikacin, CIP = Ciprofloxacin, CTX = Cefotaxime, CAZ = Ceftazidime, MRP = Meropenem, PB = Polymyxin B, TGC = Tigecycline, GEN = Gentamycin, TOB = Tobramycin, VA = Vancomycin, LZ = Linezolid, TEI = Teicoplanin, AZ = Azithromycin, CX = Cefoxitin, CD = Clindamycin, E = Erythromycin.

Table 4, Summarizes the Bacteriological Profile and Antimicrobial Susceptibility Profile of Late Onset VAP. As Compared to Early Onset VAP, the Isolates from Late Onset VAP Showed High Resistance to Ceftazidime

Antimicrobial agent	Acinetobacter baumannii (n=4)	Pseudomonas aeruginosa (n=3)	Klebsiella pneumoniae (n=4)	E. coli (n=3)	S. aureus (n = 3)	CoNS (n = 2)
Amp	0(0%)	0(0%)	2(50%)	2(66%)	-	-
PIT	2(50%)	2(66%)	2(50%)	1(33%)	-	-
AK	2 (50%)	1(33%)	3(75%)	1(33%)	-	-
CIP	3(75%)	2(66%)	4(100%)	3(100%)	3(100%)	0(0%)
CTX	3(75%)	2(66%)	3(75%)	2(66%)	-	-
CAZ	3(75%)	2(66%)	3(75%)	2(66%)	-	-
MRP	1(25%)	1(33%)	2(50%)	1(33%)	-	-
PB	4 (100%)	3 (100%)	4 (100%)	3 (100%)	-	-
TGC	2(50%)	1(33%)	2(50%)	2(66%)	0(0%)	0(0%)

GEN	2(50%)	1(33%)	3(75%)	1(33%)	1(33%)	0(0%)
TOB	2(50%)	1(33%)	3(75%)	1(33%)	-	-
COT	-	-	-	-	3(100%)	0(0%)
VA	-	-	-	-	0(0%)	0(0%)
LZ	-	-	-	-	0(0%)	0(0%)
AZ	-	-	-	-	2(66%)	1(50%)
CX	-	-	-	-	3(100)	2(100)
TEI	-	-	-	-	0(0%)	0(0%)
E	-	-	-	-	3(100%)	2(100%)
CD	-	-	-	-	3(100%)	2(100%)
P	-	-	-	-	3(100%)	2(100%)
TE	-	-	-	-	2(66)	0(0%)

Amp = Ampicillin, P = Penicillin, PIT = Piperacillin-Tazobactam, AK = Amikacin, CIP = Ciprofloxacin, CTX = Cefotaxime, CAZ = Ceftazidime, MRP = Meropenem, PB = Polymyxin B, TGC = Tigecycline, GEN = Gentamycin, TOB = Tobramycin, VA = Vancomycin, LZ = Linezolid, TEI = Teicoplanin, AZ = Azithromycin, CX = Cefoxitin, CD = Clindamycin, E = Erythromycin.

DISCUSSION

VAP, a type of HAI that involves the lung parenchyma, occurs after 48 hrs of tracheal intubation and mechanical ventilation.[9] It is reported in approximately 20–36% of critically ill patients and the rate of incidence across the world is known to vary between 2 to 16 episodes/1000 ventilator days. Incidences rates are influenced by several factors including diagnostic criteria, compliance with care bundles and other infection prevention and control policies, patient population and geographical location. [10]

The principal factor for the pathogenesis of VAP is reported to be due to aspiration of oropharyngeal pathogens and the leakage of secretions containing bacteria around the endotracheal tube. The bacteriological approach for the management of VAP avoids the problem of over treatment by separating colonizers from infecting pathogens. Quantitative cultures of endotracheal aspirate or bronchoalveolar lavage is recommended by the American Thoracic Society for confirmation of VAP.[11]

When bacteriological profile of confirmed VAP cases was studied it was noted that majority of cases are associated with gram negative organisms. This observation is accordance to that of Chawla et al who reported that 87% of patients with VAP were infected with gram negative bacilli [12].

Among the Gram-negative bacilli, non-fermenters are the predominant pathogens causing VAP in our ICU. In this study the most frequently isolated organisms in VAP patients were Acinetobacter baumannii, Pseudomonas aeruginosa, Klebsiella pneumoniae, E. coli and S. aureus. Similar result found in the study by Craven et al.[13]

The common organisms causing early onset VAP are Acinetobacter baumanii (10.34%), Pseudomonas aeruginosa (6.90%), Citrobacter freundii (6.90%), Klebsiella pneumoniae (3.45%), E.coli (3.45%) and Methicillin Sensitive Staphylococcus aureus (3.45%). Similar result is found in the study by Dey et al from Manipal where the commonest organism causing both early and late onset VAP was Acinetobacter species (48.94%) followed by P. aeruginosa (25.53%) [14]

In case of the late onset VAP organisms are Acinetobacter baumannii (13.79%), Pseudomonas aeruginosa (10.34%), Klebsiella pneumoniae (13.79%), E. coli (10.34%), Methicillin Resistant Staphylococcus aureus (10.34%), and Methicillin Resistant Coagulase Negative Staphycoccus (6.90%). Most common isolate in my study is Acinetobacter baumanii followed by Pseudomonas aeruginosa. Other studies showed similar findings. [15,16,17]

Antimicrobial susceptibility testing reflected a vast variation in susceptibility profile of isolates from late and early onset od VAP. 62.07% of these VAP pathogens were resistant to commonly used drugs. In early onset VAP 5(50%) pathogens were MDR and in late onset VAP 13(68.42%) pathogens were MDR. This study is in contrast with a study conducted by Joseph et al where 78.7% of VAP pathogens were multi drug resistant. [18]

CONCLUSION

Mechanically ventilated patients are at high risk of developing ventilator associated pneumonia (VAP), as 29% of patients developed VAP in this study. Late onset VAP is more common than early onset VAP. Acinetbacter baumannii is the commonest isolate from VAP cases. There is great variation antimicrobial susceptibility pattern of isolates from late onset VAP and early onset VAP. Hence it is necessary to understand the bacteriological profile and their susceptibility for designing the guidelines for prophylaxis and treatment of VAP cases.

REFERENCES

1. Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care–associated infection and criteria for specific types of infections in the acute care setting. American journal of infection control. 2008; 36(5):309-32.
2. Haque M, Sartelli M, McKimm J, Bakar MA. Health care-associated infections–an overview. Infection and drug resistance. 2018; 11:2321-33.
3. Rajesh E, Katragadda R, Ramani CP. Bacteriological profile and antimicrobial resistance pattern of ventilator associated pneumonia in tertiary care hospital. Indian J Microbiol Res. 2021;8(3):191-95.
4. Farag AM, Tawfick MM, Abozeed MY, Shaban EA, Abo-Shadi MA. Microbiological profile of ventilator-associated pneumonia among intensive care unit patients in tertiary Egyptian hospitals. The Journal of Infection in Developing Countries. 2020 Feb 29;14(02):153-61.
5. Chi S, Kim T, Park C, Yu J, Lee B, Lee H. Bacterial pathogens of ventilator associated pneumonia in a tertiary referral hospital. Tuberc Respir Dis (Seoul). 2012; 73(1):32-7.
6. Thakur HK, Tarai B, Bhargava A, Soni P, Ojha AK, Kancharla S, et al. Comprehensive Analysis of Etiological Agents and Drug Resistance Patterns in Ventilator-Associated Pneumonia. Microbiology Research. 2025;16(7):152.
7. Fernández-Martínez NF, Rivera-Izquierdo M, Ortiz-González-Serna R, Martínez-Ruiz V, Lardelli-Claret P, Aginagalde-Llorente AH et al. Healthcare-associated infections by multidrug-resistant bacteria in Andalusia, Spain, 2014 to 2021. Euro Surveill. 2023 Sep;28(39):2200805.
8. Chastre J, Fagon J. Ventilator-associated Pneumonia. Am J Respir Crit Care Med. 2002;165:867–903.
9. Dey A, Bairy I. Incidence of multidrug-resistant organisms causing ventilator associated pneumonia in a tertiary care hospital: A nine months' prospective study. Ann Thorac Med. 2007; 2:52–7.
10. Torres A, Niederman MS, Chastre J, Ewig S, Fernandez-Vandellos P, Hanberger H, Kollef M, et al. International ERS/ESICM/ESCMID/ALAT guidelines for the management of hospital-acquired pneumonia and ventilator-associated pneumonia: guidelines for the management of hospital-acquired pneumonia (HAP)/ventilator-associated pneumonia (VAP) of the European Respiratory Society (ERS), European Society of Intensive Care Medicine (ESICM), European Society of Clinical Microbiology and Infectious Diseases (ESCMID) and Asociación Latinoamericana del Tórax (ALAT). European Respiratory Journal. 2017;50: 1700582.
11. Ekren PK, Ranzani OT, Ceccato A, Li Bassi G, Muñoz Conejero E, Ferrer M, et al. Evaluation of the 2016 Infectious Diseases Society of America/American Thoracic Society guideline criteria for risk of multidrug-resistant pathogens in patients with hospital-acquired and ventilator-associated pneumonia in the ICU. American journal of respiratory and critical care medicine. 2018 Mar 15;197(6):826-30.
12. Chawla R. Epidemiology, etiology, and diagnosis of hospital-acquired pneumonia and ventilator-associated pneumonia in Asian countries. Am J Infect Control. 2008;36(4 SUPPL.):S93–100.
13. Craven DE. Epidemiology of Ventilator Associated Pneumonia. CHEST J. 2000;117(4):186S-187S.
14. Dey A, Bairy I. Incidence of multidrug-resistant organisms causing ventilator-associated pneumonia in a tertiary care hospital : A nine months' prospective study. Ann Thorac Med. 2007;2(2):52–7.
15. Ahmad H, Sadiq A, Bhatti HW, Bhatti AA, Tameez-ud-din A, Ibrahim A, et al. Bacteriological Profile and Antibiogram of Cultures Isolated from Tracheal Secretions. Cureus. 2019; 21:11.
16. George P SA. Antimicrobial sensitivity pattern among organisms which were isolated from the endotracheal aspirates of patients with ventilator associated pneumonia. J Clin Diagn Res. 2010;4:3397-401.
17. Gowda RS, Sowmya GS, Pavithra N, Raghavendra Rao M, Krishna KM, Satya SB. Bacteriological profile of endotracheal aspirates and their antibiotic susceptibility pattern. J Pure Appl Microbiol. 2018;12(4):2283–7.
18. Joseph NM, Sistla S, Dutta TK, Badhe AS, Parija SC. Ventilator-associated pneumonia in a tertiary care hospital in India: Incidence and risk factors. J Infect Dev Ctries. 2009;3(10):771–7.