



Study of Microbiological Pattern and Antimicrobial Resistance Pattern in Patients With Ventilator Associated pneumonia at a tertiary hospital

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Abstract

Ventilator associated pneumonia (VAP) occurs frequently and is associated with significant morbidity in critically ill patients. A knowledge of antibiotic susceptibility pattern will avoid irrational use of antibiotics in order to control the spread of infection and for proper management of VAP. Present study was aimed to study microbiological pattern and antimicrobial resistance pattern in patients with ventilator associated pneumonia at a tertiary hospital. Material and Present study was hospital based, prospective, observational study, conducted in patients >18 years, of either gender, who were on mechanical ventilation for more than 48 hours in ICU, who had Clinical Pulmonary Infection Score (CPIS) values (>6) including fever greater than 38°C, leucocytosis, oxygenation, progressive radiographic infiltrate and tracheal aspirate culture result. In present study, 153 clinically suspected patients of VAP were studied. Male to female ratio was found to be 1.55:1. Maximum number of cases put on mechanical ventilation had sepsis (37.90%) followed by miscellaneous conditions (22.87%), encephalopathy/ hemiplegia / hemiparesis (9.15%), head injury (7.84%), organo-phosphorous poisoning (7.19%), diabetic complications (5.88%). Out of 153 clinically suspected cases, 59 were found culture positive VAP. Out of 21 early onset VAP cases maximum organisms isolated were Pseudomonas aeruginosa (30.43%) followed by Acinetobacter species (17.39%), Klebsiella pneumoniae (17.39%), Escherichia coli 3 (13.04%) and Enterobacter species (4.35%). Among the Gram positive cocci, 2 (08.70%) isolates Methicillin Sensitive Staphylococcus aureus and Enterococcus faecalis each were isolated. Out of 38 late onset VAP cases maximum organisms isolated were of Pseudomonas aeruginosa (26.19 %) followed by Acinetobacter species (23.81%), Klebsiella pneumoniae (19.05%), Escherichia coli (14.28%). Ventilator-associated pneumonia complicates the prognosis of patients receiving mechanical ventilation.

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Key Words

Ventilator-associated pneumonia, mechanical Ventilation, acinetobacter antibiotic susceptibility patterns

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INTRODUCTION

Ventilator associated pneumonia (VAP) is defined as pneumonia that occurs 48 hours or more after endotracheal intubation or tracheostomy, caused by infectious agents not present or incubating at the time mechanical ventilation was started^[1]. Ventilator associated pneumonia (VAP) has been reported to be the most serious health care associated infection (HCAI) particularly in the intensive care unit (ICU)^[2]. Patients admitted in ICUs have an increased susceptibility to infection because of decreased mobility and increased use of invasive devices. It influences the length of stay, cost of treatment and patient mortality^[3].

Diagnosing VAP requires a high clinical suspicion combined with bed side examination, radiographic examination and microbiological analysis of respiratory secretions. ATS guidelines recommended that endotracheal aspirate (ETA) or bronchoscopic aspiration from the infected lungs' segments should be sent for quantitative/semi-quantitative culture and antimicrobial susceptibility testing for the precise diagnosis and proper antimicrobial therapy^[4].

The aetiologic agents of VAP include some of the common hospital pathogens such as Pseudomonas spp., Acinetobacter and other non-fermenters, members of the Enterobacteriaceae family, as well as Gram-positive pathogens such as Staphylococci and the fungal agent Candida^[5,6]. Emergence of drug resistance against the microorganism causing VAP is a serious concern in most of the ICUs. A knowledge of antibiotic susceptibility pattern will avoid its irrational use in order to control the spread of infection and for proper management of VAP. Present study was aimed to study microbiological pattern and antimicrobial resistance pattern in patients with ventilator associated pneumonia at a tertiary hospital.

MATERIAL AND METHODS

Present study was hospital based, prospective, observational study, conducted in department of microbiology in the ICU of Department of Critical Care Medicine, at GMC and hospital, Nagpur, India. Study duration was of 2 year (January 2022-December 2023). Study was approved by institutional ethical committee.

Inclusion Criteria:

 Patients >18 years, of either gender, who were on mechanical ventilation for at least 5 days in ICU, who had Clinical Pulmonary Infection Score (CPIS) values (>6) including fever greater than 38°C, leucocytosis, oxygenation, progressive radiographic infiltrate and tracheal aspirate culture result

Exclusion Criteria:

- Patients with extra pulmonary infection sources, surgical history
- Patients with any previous antibiotic therapy <48 hour before the study

Mechanically ventilated patients who developed VAP during the study period were included in the study.

Under strict aseptic precautions respiratory secretions such as endotracheal aspiration (ETA) were collected from patients on ventilation. The first sample of ETA was collected after 48 hours of intubation and subsequently samples were collected every 24 hours to see significant colonization.

Blood samples for blood culture were collected and processed as per protocol using standard microbiological techniques. Bacteriological media such as Blood agar, Chocolate agar, MacConkey agar by semi-quantitative culture technique by using standard bacteriological loop.

Significant growth was considered when >105 colonies for ETA.7 Bacterial isolates were identified based on colony characters and biochemical parameters. The antibiotic susceptibility profile was determined for these isolates by Kirby-Bauer disc diffusion method as per Clinical and laboratory Standards Institute (CLSI) 2022 guidelines.

The antimicrobial susceptibility pattern was tested using filter paper discs containing a specific concentration of antimicrobial drugs. Accordingly, susceptibility of the isolates to following antibiotics: amikacin (30 µg/disk), trimethoprim/sulfamethoxazole $(1.25/23.75 \mu g/disk)$, cefepime (30 $\mu g/disk$), ceftriaxone (30 µg/disk), piperacillin/tazobactam (100/10 μg), ciprofloxacin (5 mg), meropenem (10 μg/disk), ceftazidime (30 μ g/disk) ampicillin-sulbactam (10/10 µg) (Mast Co., Darmstadt, Germany) were examined. In addition, Minimum Inhibitory Concentrations (MICs) were determined by the E-test method according to the manufacturer's guidelines for colistin against A. baumannii, Klebsiella pneumoniae (K. pneumoniae), and P. aeruginosa (AB Biodisk). The MIC was read where inhibition of growth intersected the E-test strip. When small colonies grew within the zone of inhibition or a haze of growth occurred around MIC end points, the highest MIC intersect was recorded.

Data was collected and compiled using Microsoft Excel, analysed using SPSS 23.0 version. Frequency, percentage, means and standard deviations (SD) was calculated for the continuous variables, while ratios and proportions were calculated for the categorical variables. Difference of proportions between qualitative variables were tested using chi- square test

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or Fisher exact test as applicable. P<0.5 was considered as statistically significant.

RESULTS AND DISCUSSIONS

In present study, 153 clinically suspected patients of VAP were studied. Male to female ratio was found to be 1.55:1. Maximum 64 (83.70%) of these patients were seen in above 60 years of age group. Maximum males 46 (49.46%) were from above 60 years, maximum females 20 (33.33) were from 21-40 years.

Radiologically, majority 63 (41.18%) had cavitations followed by 47 (30.72%) cases with new or progressive and persistent infiltrate while the remaining 43(28.10%) cases had consolidation as the radiological feature.

Endotracheal aspirate was found culture positive with cut-off value of 105 CFU/ml by semi-quantitative method in 50 (32.68%) cases. Whereas those below cut-off values <105 CFU/ml were 30 (19.61%) and with no growth in endotracheal aspirate culture were 73 (47.71%) cases.

Out of 153 clinically suspected VAP patients, blood culture was found positive in 23 (15.03%) cases. Among the 23 positive blood culture, 4 (17.39%) shown 105 CFU/ml in ETA culture while 9 (39.13%) shown <105 CFU/ml and rest 10 (43.48%) positive blood culture shown no growth on ETA culture.

Out of total 59 culture confirmed VAP positive cases, mono-microbial (one bacterial species in ETA) growth was seen in 53 (89.83%) cases whereas poly-microbial (two / more bacterial species in ETA) growth was seen in 6 (10.17%) cases, 21 (35.59%) were categorized in the early-onset group and the remaining 38 (64.41%) in the late-onset group

Out of 59 culture confirmed VAP positive cases majority were males 38 (64.41%) and remaining 21 (35.59%) were females. Majority of culture confirmed VAP positive cases.

New or progressive and persistent infiltrate was noted in 11 (52.38%) early onset VAP cases and 6 (15.78%) of late onset VAP cases. New or progressive and persistent consolidation was noted in 7 (33.33%) of early onset VAP cases and 9 (23.68%) of late onset VAP cases. New or progressive and persistent cavitation was noted in 3 (14.29%) of early onset VAP cases whereas 23 (60.52%) of late onset VAP cases, difference was highly statistically significant (p=0.01).

Maximum number of cases put on mechanical ventilation had sepsis (37.90%) followed by miscellaneous conditions (22.87%), encephalopathy/hemiplegia/hemiparesis (9.15%), head injury (7.84%), organo-phosphorous poisoning (7.19%), diabetic complications (5.88%).

Out of 21 early onset VAP cases maximum organisms isolated were Pseudomonas aeruginosa

(30.43%) followed by Acinetobacter species (17.39%), Klebsiella pneumoniae (17.39%), Escherichia coli 3 (13.04%) and Enterobacter species (4.35%). Among the gram positive cocci, 2 (08.70%) isolates Methicillin Sensitive Staphylococcus aureus and Enterococcus faecalis each were isolated. Out of 38 late onset VAP cases maximum organisms isolated were of Pseudomonas aeruginosa (26.19 %) followed by Acinetobacter species (23.81%), Klebsiella pneumoniae (19.05%), Escherichia coli (14.28%).

Out of 6 Staphylococcus aureus, 1 (16.67%) isolate was resistant to Penicillin and cefoxitin both. 3 (50%) isolates were resistant to gentamicin whereas 2 (33.33%) isolates were resistant to doxycycline. All the S. aureus isolates were 100% sensitive to erythromycin, clindamycin, linezolid and vancomycin. All the 3 Enterococcus faecalis isolates were 100% sensitive to penicillin, erythromycin, doxycycline, gentamicin, linezolid and vancomycin.

Out of 12 Klebsiella pneumoniae isolates, all were resistant to ampicillin and cefoxitin, followed by 6 (50%) isolates resistant to PIT. Resistance to ceftazidime was found in 5 (71.40%) isolates whereas 3 (25%) isolates were resistant to ceftriaxone. 2 (28.60%) isolates were resistant to cefepime. 1 (08.33%) isolate was resistant to aztreonam. Among amino glycosides maximum resistance i.e. 3 (25.00%) isolates were resistant to gentamicin and amikacin each followed by 1 (08.33%) isolate was resistant to both netilmicin and tobramycin each. 1 (08.33%) isolate was resistant to both imipenem and meropenem.

Out of 9 Escherichia coli isolates, maximum i.e. 5 (55.56%) isolates were resistant to ampicillin, cefoxitin, ceftriaxone and PIT each followed by 3 (33.33%) isolates resistant to ceftazidime. 2 (22.22%) isolates were resistant to both cefepime and aztreonam. Among amino glycosides, maximum resistance i.e. 6 (66.67%) isolates were resistant to gentamicin followed by 5 (55.56%) isolates which were resistant to amikacin while 2 (22.22%) isolates were resistant to both tobramycin and netilmicin. 2 (22.22%) isolates were resistant to meropenem and imipenem each.

Out of 2 Enterobacter species, all (100%) were resistant to ampicillin, cefepime, cefoxitin, ceftriaxone, ceftazidime aztreonam and PIT. Both the strains were sensitive to imipenem and meropenem. Among amino glycosides, all were resistant to gentamicin whereas 1 (50%) isolate was resistant to amikacin. All were sensitive to both netilmicin and tobramycin.

Citrobacter freundii was resistant to ampicillin, cefepime, cefoxitin, ceftriaxone, ceftazidime, aztreonam and PIT. All were sensitive to imipenem and meropenem. Among amino glycosides, all isolate was resistant to gentamicin and amikacin both while all

Table	1.	Ago and	aandar	distribution

Age group (years)	Male (%)	Female (%)	Total (%)
12-20	3 (3.23)	5(8.33)	8(5.28)
21-40	15(16.13)	20(33.33)	35(22.88)
41-60	29(31.82)	17(28.33)	46(30.07)
>60	46(49.46)	18(30)	64(41.83)
Total	93 (60.78)	60 (39.21)	153 (100)

Table 2: Distribution according to radiological features

Radiological features	No. of Patients	Percentage
Infiltrate	47	30.72
Consolidation	43	28.10
Cavitations	63	41.18

Table 3: General characteristics of endotracheal aspirate culture (ETA)

ETA culture (n=153)	No. of patients	Percentage
>105 CFU/ml	50	32.68
105 CFU/ml	30	19.61
No growth	73	47.71

Table 4: Comparison of positive blood cultures with ETA culture

ETA culture	Blood culture positive n=23 (%)	Blood culture negative n=130 (%)
105	04 (17.39)	46 (35.38)
<u><</u> 105	09 (39.13)	40 (30.77%)
No growth	10 (43.48)	44 (33.85)

Table 5: Culture confirmed VAP cases and onset of VAP

Characteristic	No. of patients (n=59)	Percentage
Culture confirmed VAP cases		_
Pure growth (monomicrobial)	53	89.83
Mixed growth (polymicrobial)	06	10.17
Onset of VAP		
Early (48-96 hours)	21	35.59
Late (>96 hours)	38	64.41

Table 6: Age and sex distribution in culture confirmed VAP positive cases (n=59)

Age group (years)	Male (%)	Female (%)	Total (%)
12-20	02 (05.27)	01 (04.76)	03 (05.08)
21-40	10 (26.31)	08 (38.10)	18 (30.51)
41-60	18 (47.37)	08 (38.10)	26 (44.07)
>60	08 (21.05)	04 (19.04)	12 (20.34)
Total	38 (64.41)	21 (35.59)	59 (100)

Table 7: Radiological features in culture confirmed VAP positive cases (n=59)

New or progressive and persistent radiological feature	Early-onset VAP (%)	Late-onset VAP (%)	Total (%)	X2=13.31 DF=2)		
Infiltrate	11 (52.38)	06 (15.78)	17 (28.81)	P=0.01		
Consolidation	07 (33.33)	09 (23.68)	16 (27.11)	(HS)		
Cavitations	03 (14.29)	23 (60.52)	26 (44.06)			
Total	21 (35.59)	38 (64.41)	59 (100)			

Table 8: Distribution according to underlying clinical spectrum of disease

	Culture confirmed VAP	Culture negative VAP	Total	
Clinical Spectrum	n = 59 (%)	n = 94 (%)	n = 153 (%)	p-value
Sepsis	26 (44.06)	32 (34.04)	58 (37.90)	0.13
Acute renal failure	02 (03.38)	06 (06.38)	08 (05.22)	0.84
Burns	00 (00)	02 (02.12)	02 (01.31)	0.37
Malignancies	01 (01.69)	03 (03.19)	04 (02.61)	0.78
Diabetic complications	04 (06.77)	05 (05.31)	09 (05.88)	0.82
Encephalopathy/ Hemiplegia/hemiparesis	02 (03.38)	12 (12.76)	14 (09.15)	0.56
Enteric fever	00 (00)	02 (02.12)	02 (01.31)	0.37
Epidermolysis bullosa	00 (00)	02 (02.12)	02 (01.31)	0.37
Fractures	00 (00)	03 (03.19)	03 (01.96)	0.26
Head injury/ Road Traffic Accident (RTA)	06 (10.16)	06 (06.38)	12 (07.84)	0.81
Liver disease	00 (00)	02 (02.12)	02 (01.31)	0.37
Myocardial infarction	02 (03.38)	06 (06.38)	08 (05.22)	0.84
Organophosphorus (OP) poisoning	08 (13.55)	03 (03.19)	11 (07.19)	0.06
Pancreatitis	02 (03.38)	06 (06.38)	08 (05.22)	0.84
Miscellaneous*	06 (10.16)	29 (30.85)	35 (22.87)	0.10

Table 9: Distribution of organisms according to onset of VAP (n=65)

Organism	Early-onset VAP n = 21 (%)	Late-onset VAP n = 38 (%)	Total (%)
Pseudomonas aeruginosa	07 (30.43)	11 (26.19)	18 (27.69)
Acinetobacter spp	04 (17.39)	10 (23.81)	14 (21.54)
Klebsiella pneumoniae	04 (17.39)	08 (19.05)	12 (18.46)
Escherichia coli	03 (13.04)	06 (14.28)	09 (13.85)
Enterobacter spp	01 (04.35)	01 (02.38)	02 (03.08)
Methicillin Sensitive Staphylococcus aureus (MSSA)	02 (08.70)	03 (07.14)	05 (07.69)
Methicillin Resistant Staphylococcus aureus (MRSA)	00 (00)	01 (02.38)	01(01.54)
Enterococcus faecalis	02 (08.70)	01 (02.38)	03 (04.61)
Citrobacter freundii	00 (00)	01 (02.38)	01 (01.54)

Table 10: Antimicrobial resistance pattern of gram positive isolates

Antibiotic discs (concentration)	Staphylococcus aureus n = 06 (%)	Enterococcus faecalis n = 03 (%)
Penicillin (10 units)	01 (16.67)	00 (00)
Cefoxitin (30 μg)	01 (16.67)	-
Erythromycin (15 μg)	00 (00)	00 (00)
Clindamycin (2 μg)	00 (00)	-
Doxycycline (30 μg)	02 (33.33)	00 (00)
Gentamicin (10 μg)	03 (50.00)	00 (00)
Vancomycin (30 μg)	00 (00)	00 (00)
Linezolid (30 ug)	00 (00)	00 (00)

Table 11: Antimicrobial resistance pattern among Enterobacteriaceae isolates

Antibiotic	Klebn = 12 (%)	E. colin = 9 (%)	Citrobacter n = 1 (%))	Enterobacter n = 2(%)	Total n = 24 (%)
Ampicillin (10μg)	12 (100)	05 (55.56)	01 (100)	02 (100)	20 (83.33)
Amikacin (30μg)	03 (25.0)	05 (55.56)	01 (100)	01 (50)	10 (41.67%)
Aztreonam (10μg)	01 (8.33)	02 (22.22)	01 (100)	02 (100)	06 (25 %)
Cefoxitin (30µg)	12 (100)	05 (55.56)	01 (100)	02 (100)	20 (83.33)
Ceftriaxone (30µg)	03 (25.0)	05 (55.56)	01 (100)	02 (100)	11 (45.83)
Ceftazidime (30µg)	05 (71.4)	03 (33.33)	01 (100)	02 (100)	11 (45.83)
Cefepime (30µg)	02 (28.6)	02 (22.22)	01 (100)	02 (100)	07 (29.17)
Ciprofloxacin (30µg)	07 (58.3)	05 (55.56)	01 (100)	02 (100)	15 (62.50)
Gentamicin (10μg)	03 (25.0)	06 (66.67)	01 (100)	02 (100)	12 (50 %)
Imipenem (10μg)	01 (8.33)	02 (22.22)	00 (00)	00 (00)	03 (12.50)
Meropenem (10μg)	01 (8.33)	02 (22.22)	00 (00)	00 (00)	03 (12.50)
Netilmicin (30μg)	01 (8.33)	02 (22.22)	00 (00)	00 (00)	03 (12.50)
Piperacillin-Tazobactam (PIT) (100/10μg)	06(50)	05 (55.56)	01(100)	02(100)	14 (58.33)
Tobramycin (10μg)	01 (8.33)	02 (22.22)	00 (00)	00 (00)	03 (12.50)
Colistin (10μg)	01 (8.33)	02 (22.22)	00 (00)	-	03 (12.50)

Table 12: Antimicrobial resistance pattern in non-fermenter isolates in VAP positive cases

Antibiotic	Pseudomonas aeruginosan = 18 (%)	Acinetobacter sppn = 14 (%)	Total n = 32 (%)
Amikacin (30µg)	07 (38.89)	09 (64.3)	16 (50.00)
Aztreonam (30µg)	11 (61.11)	11 (78.6)	22 (68.75)
Cefoxitin (30µg)	12 (66.67)	13 (92.9)	25 (78.13)
Ceftazidime (30µg)	07 (38.89)	13 (92.9)	20 (62.50)
Cefepime (30µg)	09 (50.00)	12 (85.7)	21 (65.63)
Ciprofloxacin (5µg)	15 (83.33)	13 (92.9)	28 (87.50)
Gentamicin (10µg)	14 (77.78)	11 (78.6)	25 (78.13)
Imipenem (10µg)	06 (33.33)	09 (64.3)	15 (46.88)
Meropenem (10μg)	08 (44.44)	10 (71.4)	18 (56.25)
Netilmicin (30µg)	05 (27.78)	10 (71.4)	15 (46.88)
Piperacillin-Tazobactam (PIT) (100/10μg)	08 (44.44)	11 (78.6)	19 (59.37)
Tobramycin (10μg)	11 (61.11)	09 (64.3)	20 (62.50)

were sensitive to both netilmicin and tobramycin. All were sensitive to colistin.

Thus, out of the total 24 Enterobacteriaceae, maximum 20 (83.33) isolates were resistant to ampicillin and cefoxitin each followed by 14 (58.33%) isolates resistant to PIT. 11 (45.83%) isolates were resistant to ceftazidime and ceftriaxone each whereas 7 (29.17%) isolates were resistant to cefepime. Among the amino glycosides maximum 12 (50%) isolates were resistant to Gentamicin followed by 10 (41.67%) isolates were resistant to amikacin whereas 3 (12.50%) isolates were resistant to netilmicin and tobramycin each. 3 (12.50%) isolates were found resistant to imipenem and meropenem each. 3 (12.50%) isolates were resistant to colistin.

Out of 18 Pseudomonas aeruginosa showed maximum i.e., 12 (66.67%) resistance to cefoxitin, followed by 11 (61.11%) to aztreonam. 9 (50%) were resistant to cefepime whereas 8 (44.44%) were resistant to PIT. 7 (38.89%) isolates were resistant to ceftazidime. Among the amino glycosides, 14 (77.78%) isolates showed resistance to gentamicin, 11 (61.11%) isolates to tobramycin, 7 (38.89%) isolates to amikacin, and 5 (27.78%) to netilmicin. Imipenem showed a resistance of 44.44%. All the isolates were sensitive to

colistin, polymyxin B and tigecycline.

Out of 14 Acinetobacter species showed maximum i.e., 13 (92.90%) isolates resistance to both cefoxitin and ceftazidime, followed by 12 (85.7%) isolates resistant to cefepime while 11 (78.60%) isolates resistant to aztreonam and PIT each. Among the amino glycosides, 11 (78.60%) isolates showed resistance to gentamicin, 10 (71.4%) isolates to netilmicin while 9 (64.30%) isolates to both tobramycin and amikacin each. Imipenem showed a resistance of 64.3% while meropenem showed a resistance of 71.4%. All the isolates were sensitive to tigecycline.

VAP occurs frequently and is associated with significant morbidity in critically ill patients. Risk factors of VAP include oropharyngeal colonization, trauma, surgery, immunosuppression, old age, urgent intubation, prolonged admission in ICU, sedative drugs steroids usage and previous hospitalization^[8].

Patients with mechanical ventilation have an increased risk for respiratory tract infection because the tube which has been inserted in trachea reduces the clearance of bacteria and increases the leakage of secretion around the cuff of tube and disable the ciliary tract by damaging it^[9,10]. VAP can be categorized into early onset and late onset based on the occurrence within or after 4 days respectively. Early onset VAP is

usually caused by pathogens that are sensitive to various antimicrobial agents, whereas late onset VAP is mostly caused by multi-drug resistant (MDR) pathogens^[11,12].

Pneumonia is usually mild or low in severity if it occurs in the early period of invasive ventilation and the organisms are most responsive to the antibiotics administered Whereas after a few days (late-onset), pneumonia is more severe in its course, with fewer organisms responding to antibiotics and increased rate of morbidity and mortality among those with late-onset infection^[13].

Our study findings were similar to findings in study done by Anuradha De *et al.*, [14]

Incidence of VAP as 40.8%, out of which majority had late-onset VAP. In our study VAP incidence is 38.56% and majority had Late-onset VAP. Diabetes mellitus, advancing age (>60 years) and Chronic Obstructive Pulmonary Disease were the important risk factors associated with VAP. The most frequently isolated microorganism was Acinetobacter species (70.37%), followed by Pseudomonas aeruginosa (14.81%) and Klebsiella pneumoniae (5.56%). Blood culture results were positive for 47.16% cases of VAP of which 76% showed bacteremia of pulmonary origin. Acinetobacter species (33.96%) was the most common isolate from blood. In our study similar findings were noted that is Non-fermenter were frequently isolated in both ETA culture and Blood culture positive VAP

Ranjan N et al., [15] noted that majority of bacterial isolates causing VAP were found to be Gram negative bacilli finding similar to the present study. Acinetobacter spp (34.28%) followed by Pseudomonas aeruginosa (25.71%). Other gram negative bacilli isolated were Klebsiella pneumoniae, Citrobacter freundii, Enterobacter spp and Escherichia coli.

Similar Microbiological profile of VAP was found by Sangale A *et al.*, [16] studied 1,074 patients: 710 (66.10%) men and 364 (33.90%) women. A total of 827 bacterial isolates were obtained with 780 (94.32%) Gram-negative organisms and 47 (5.68%) Gram-positive organisms, of which Acinetobacter baumannii (38.7%), Pseudomonas aeruginosa (17.5%), and Klebsiella pneumoniae (16.6%) were the commonest.

In study by Rajesh E *et al.*,^[17] similar findings was noted - among the isolates, the most common were Klebsiella pneumoniae (31.31%) and Acinetobacter baumanii (31.31%). These were followed by Pseudomonas aeruginosa (22.22%), Klebsiella oxytoca (7.07%), Escherichia coli (3.03%) and Proteus mirabilis (3.03%) and Staphylococcus aureus (2.02%).

Aggressive surveillance is vital in understanding local factors leading to VAP and the micro biologic milieu of a given unit. Judicious antibiotic usage is essential as resistant organisms continue to plague ICUs and critically ill patients. Simple and effective

preventive measures such measures might include, diligent respiratory care, hand hygiene, the elevation of the head, oral and not nasal cannulation, minimization of sedation, the institution of weaning protocols, judicious use of antibiotics, can be instituted easily and at minimal costs.

CONCLUSION

Ventilator-associated pneumonia complicates the prognosis of patients receiving mechanical ventilation. VAP is a serious problem in ICU carrying many risks for the patient live. Regimens of empirical treatment should take in consideration the update in the bacterial etiology and antibiotic susceptibility patterns of VAP.

REFERENCES

- ATC. and IDSA., 2005. Guidelines for the management of adults with hospital-acquired, ventilator-associated and healthcare-associated pneumonia. Am. J. Respir. Critic. Care Med., 171: 388-416.
- Xie, D.S., W. Xiong, R.P. Lai, L. Liu and X.M. Gan et al., 2011. Ventilator-associated pneumonia in intensive care units in hubei province, China: A multicentre prospective cohort survey. J. Hosp. Infec., 78: 284-288.
- Alp, E. and A. Voss, 2006. Ventilator associated pneumonia and infection control. Ann. Clin. Microbiol. Antimicrob., Vol. 5 .10.1186/1476-0711-5-7.
- Ali, S., K. Waheed and Z.H. Iqbal, 2015. Microbiological pattern of ventilator-associated pneumonia. J. Ayub Med. Coll. Abbottabad, 27: 117-119.
- Bairy, I. and A. Dey, 2007. Incidence of multidrug-resistant organisms causing ventilator-associated pneumonia in a tertiary care hospital: A nine months' prospective study. Ann. Thoracic Med., 2: 52-57.
- Medell, M., M. Hart, O. Marrero, F. Espinosa, Z.M. de Oca and R. Valdés, 2012. Clinical and microbiological characterization of pneumonia in mechanically ventilated patients. Braz. J. Infect. Dis., 16: 442-447.
- Tablan, O.C., L.J. Anderson, R. Besser, C. Bridges and R. Hajjeh, 2004. Guidelines for preventing health-care-associated pneumonia, 2003: Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. MMWR. Recomm. Rep., Vol. 53.
- Maleh, P., M. Sadeghi-Haddad-Zavareh, H. Jouybari, M. Javanian and M. Shokri et al., 2018. Antimicrobial resistance pattern in ventilator-associated pneumonia in an intensive care unit of Babol, Northern Iran. J. Acute Dis., 7: 74-77.

- Ferreira, T.D., R.Y. Koto, G.F.D. Leite, G.B. Klautau and S. Nigro et al., 2016. Microbial investigation of biofilms recovered from endotracheal tubes using sonication in intensive care unit pediatric patients. Braz. J. Infect. Dis., 20: 468-475.
- Tandia, K., J.L. Wadhwani and M. Sharma, 2015. A clinical study of pattern of microbiological colonization of endotracheal tube aspirate on mechanically ventilated patients. Int. J. Sci. Res., 4: 785-787.
- 11. Hunter, J.D., 2012. Ventilator associated pneumonia. BMJ, Vol. 344 .10.1136/bmj.e3325.
- 12. Joseph, N.M., S. Sistla, T.K. Dutta, A.S. Badhe and S.C. Parija, 2009. Ventilator-associated pneumonia in a tertiary care hospital in India: Incidence and risk factors. J. Infec. Dev. Ctries., 3: 771-777.
- De, A., A. Samaddar, S. Patwegar and S. Baveja, 2018. Antibiotic susceptibility pattern of bacteria isolated from adult patients with Ventilator Associated Pneumonia (VAP) in intensive care units in a Tertiary Care Hospital. JMSCR., 6: 1104-1112.

- Ranjan, N., K. Ranjan, U. Chaudhary and D. Chaudhry, 2014. Antimicrobial resistance in bacteria causing ventilator-associated pneumonia in a tertiary care hospital: One year prospective study. Int. J. Res. Med. Sci., 2: 228-233.
- 15. Kelkar, R., A. Sangale, V. Bhat and S. Biswas, 2021. Microbiology of ventilator-associated pneumonia in a tertiary care cancer hospital. Indian J. Crit. Care Med., 25: 421-428.
- Rajesh, E., R. Katragadda and C.P. Ramani, 2021.
 Bacteriological profile and antimicrobial resistance pattern of ventilator associated pneumonia in tertiary care hospital. Indian J. Microbiol. Res., 8: 191-195
- Gatell, M.R.J., M.S. Roig, Ó.H. Vian, E.C. Santín, C.T. Duaso, I.F. Moreno and J.V. Daunis, 2012. Assessment of a training programme for the prevention of ventilator-associated pneumonia. Nurs. Crit. Care, 17: 285-292