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Microbial Profile and Antibiogram of Ventilator Associated Pneumonia at Tertiary Care Hospital U.P

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ABSTRACT

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Ventilator associated pneumonia (VAP) is a common and serious healthcare associated infection (HAI). VAP is inflammation of lung parenchyma caused by infectious agents that develops in a patient who is on a ventilator occurring 48-72 hrs or more after tracheal intubation and mechanical ventilation (MV). The risk of VAP is highest early in the course of hospital stay, and is estimated to be 3%/day during the first 5 days of ventilation, 2%/day during days 5-10 of ventilation and 1%/day after this. Aim of this study was find out the Microbial profile and antibiogram of ventilator associated pneumonia at tertiary care hospital U.P. This study was conducted in the Department of Microbiology, Rama Medical College, Hospital & Research Centre, Kanpur India from January 2016 to December 2016. A total of 100 Endotracheal aspirated samples were collected in a sterile & labelled clean dry container from the clinically suspected cases of VAP patients. Out of 100 patients 31 patients shown growth of the bacteria & considered as Ventilator Associated Pneumonia patients. And 69 patients have not shown any kind of growth. *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Enterobacter* & *Klebsiella Pneumoniae* were the most common pathogens from both early & late onset VAP. Proper monitoring and strict implementation of infection control practices is very essential to reduce VAP in hospital setting. Also the short term use of invasive devices and judicious use of antibiotics are important in preventing VAP caused by these MDR pathogens.

Introduction

Ventilator-associated pneumonia (VAP) is a lung infection that develops in a person who is

on ventilator. It is a common infectious disease that is found in intensive care unit (ICU).⁽¹⁾ A ventilator is a machine that is used to help a patient breathe by giving oxygen

through a tube placed in a patient's mouth or nose, or through a hole in the front of the neck. An infection may occur if germs enter through the tube and get into the patient's lungs parenchyma. Infectious agents that develops in a patient who is on a ventilator occurring 48-72 hrs or more after tracheal intubation and mechanical ventilation (MV).^(2,3) Intubation independently increases the risk of the developing pneumonia. It is the most common hospital associated infection accounting for 9-28% of all incubated patients.⁽⁴⁾ Ventilator associated pneumonia may be of early onset and late onset. Early onset Ventilator associated pneumonia, defined as occurring within the first four days of hospitalization, usually carry a better prognosis, and is more likely to be caused by antibiotic-sensitive bacteria. Late onset Ventilator associated pneumonia when it develops five or more days and more likely to be caused by multidrug-resistant (MDR) pathogens, and are associated with increased patient mortality and morbidity.⁽²⁾ Early-onset nosocomial pneumonia was believed due to primarily gram-negative bacteria, such as *Haemophilus influenzae*, and Methicillin-sensitive *Staphylococcus aureus* (MSSA) and *Streptococcus pneumoniae*.

For late-onset nosocomial pneumonia causative pathogens reported were higher-level antibiotic-resistant gram-negative bacteria, such as *Pseudomonas aeruginosa*, *Acinetobacter* spp and Methicillin-resistant *S. aureus* (MRSA).⁽⁵⁾ Mechanically ventilated patients are at high risk for complications and poor outcomes, including death. Ventilator-associated pneumonia (VAP), sepsis, Acute Respiratory Distress Syndrome (ARDS), pulmonary embolism, barotrauma, and pulmonary edema are among the complications that can occur in patients receiving mechanical ventilation; such complications can lead to longer duration of mechanical ventilation, longer stays in the

ICU and hospital, increased healthcare costs, and increased risk of disability and death.⁽³⁾ The risk of VAP is highest early in the course of hospital stay, and is estimated to be 3% per day during the first 5 days of ventilation, 2% per day during days between 5-10 of ventilation and 1% per day after this.⁽⁶⁾

Risk factor of VAP includes- Increases the risk of colonization by potential pathogens (e.g.- prior antibiotic therapy, contaminated ventilator circuits, decreased gastric acidity).⁽⁷⁾ Increased possibility of aspiration of pharyngeal contents in to the lower respiratory tract (e.g. intubation, decreased level of consciousness, presence of nasogastric tube).⁽⁸⁾ reduced host defence mechanism in the lung and permit overgrowth of aspirated pathogen (e.g. chronic obstructive pulmonary disease, old age, upper abdominal surgery).⁽⁹⁾ Clinical strategy for ventilator associated pneumonia is the presence of a new progressive radiographic infiltrates and the presence of at least two of three clinical features fever greater than 38 c, leukocytosis or leukopenia and purulent secretions represent the most accurate clinical criteria for starting empiric antibiotic therapy. This is the clinical strategy as defined by the American Thoracic Society (ATS) & Infectious Disease Society of America (IDSA).⁽²⁾

Materials and Methods

This Study was conducted in the Department of Microbiology, Rama Medical College, Hospital & Research Centre, Kanpur India from January 2016 to December 2016. We assessed the clinical parameters which include the history, the number of patient spent on ventilator, the timing of sample collection, clinical examination of the patient and relevant investigations. This included the blood counts, renal function tests, blood glucose, liver function tests and chest X-rays or any other relevant investigations.

Inclusion criteria

All ICU patients admitted for more than 48hrs, Patients on mechanical ventilator of all age groups & both sex were included.

Exclusion criteria

Patients already on antibiotic therapy and admitted in ICU less than 48 hrs, Patients with pneumonia prior to mechanical ventilation with Adult Respiratory, Distress Syndrome (ARDS), cavitary lung disease based on chest X-ray findings, primary lung cancer or another malignancy metastatic to the lungs and cystic fibrosis & Tuberculosis patients were excluded.

Specimen collection

A total of 100 Endotracheal aspirated samples were collected in a sterile & labelled clean dry container from the clinically suspected cases of VAP patients. Specimens were transported to the laboratory as soon as possible after collection. If delay was unavoidable, the specimens was refrigerated at 4°C to inhibit the growth of unwanted microorganisms.

Specimen processing

The Endotracheal aspirates sent to the lab were processed immediately. The samples were first subjected to Gram's staining and then quantitative cultures were performed.

The samples were inoculated on Sheep Blood agar, Chocolate agar, and MacConkey agar. All the plates were incubated for 24 to 48 hours at 37°C. Bacterial isolates were identified by their colony characteristics & morphology by gram's staining reaction and standard biochemical reactions. Antimicrobial

susceptibility testing was performed for all the isolates with positive cultures according to Kirby-Bauer's disk diffusion method following Clinical Laboratory Standard (CLSI) Guideline 2016.⁽¹⁰⁾

Results and Discussion

Total 100 patients were included in this study who were on mechanical ventilator for more than 48 hours. Out of 100 patients 31 patients shown growth of the bacteria & considered as Ventilator Associated Pneumonia patients. 69 patients have not shown any kind of growth of microorganisms. Out of 31 VAP patients 09 patients showed two types of bacteria so during the study we got total 40 isolates of bacteria. *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Enterobacter* & *Klebsiella Pneumoniae* were the most common pathogens from both early & late onset VAP. VAP is more common in men 21(67.74%) than women 10 (32.25%) in our study. The maximum cases of VAP were found in the elderly age group of 61-75 years of patients.

The administration of accurate and timely initial empirical antibiotic therapy has been shown to have a major impact on mortality from nosocomial pneumonia. Because early-onset nosocomial pneumonia is most often reported as being due to antibiotic-sensitive pathogens, while late-onset nosocomial pneumonia is frequently caused by more-resistant pathogens, guidelines recommend monotherapy with narrow-spectrum antibiotics for early-onset infections and broad-spectrum therapy for late-onset infections.⁽¹⁴⁾ The incidence of VAP in our study was 31%, which was about nearly similar to studies done by Shalini *et al.*, (35.78%)⁽¹⁵⁾ and Gadani *et al.*, (37%)⁽¹⁶⁾

Table.1 Age wise distribution of VAP cases.

Age group (years)	Total (n=100)	VAP (n=31)	%
< 15	30	01	3.3
16-30	13	02	15.3
31-45	15	03	20
46-60	18	08	44
61-70	22	15	68.1
>75	02	02	100

Table.2 Showing isolates from VAP cases.

S. No.	Isolated Organisms	No. of positive	%
1	<i>Acinetobacter baumannii</i>	16	40
2	<i>Enterobacter aerogens</i>	5	12.5
3	<i>Pseudomonas aeruginosa</i>	6	15
4	<i>Streptococcus pneumonia</i>	1	2.5
5	<i>Klebsiella pneumonia</i>	3	7.5
6	MRSA	2	5
7	<i>Citrobacter freundii</i>	2	5
8	<i>Escherichia coli</i>	1	2.5
9	<i>Candida albicans</i>	1	2.5
10	<i>Candida tropicalis</i>	1	2.5
11	<i>Candida krusei</i>	1	2.5
12	<i>Candida glabrata</i>	1	2.5

Fig.1 Bar diagram shows Age wise distribution of VAP cases

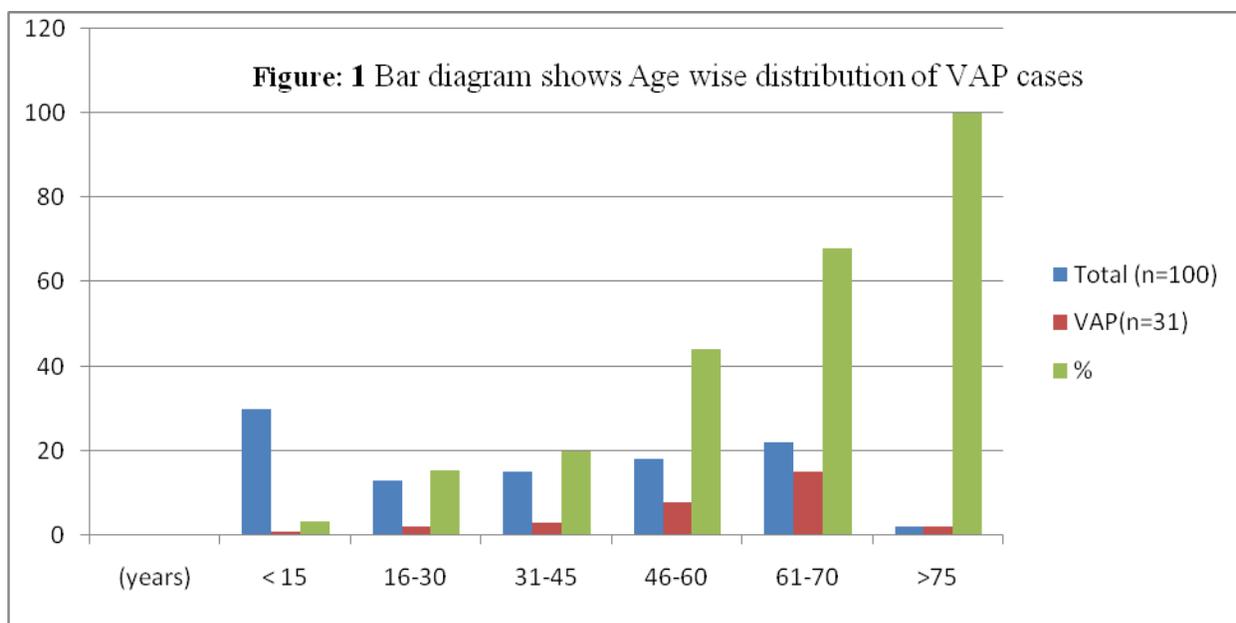


Table.3 Showing Antibiotics sensitivity Pattern of different Isolates.

	MRP	AMC	A/S	PIT	CTX	CTR	CFS	CPM	AK	GN	CIP	LE	TE	IMP	CL	PB
<i>Acinetobacter</i>	63.5	0	0	63.6	22.2	27.2	75	0	36.3	50	22.2	63.6	75	81.8	100	100
<i>Klebsiella oxytoca</i>	0	0	0	0	0	0	0	0	100	100	0	100	100	100	100	100
<i>Klebsiella pneumoniae</i>	0	0	50	66.6	50	0	33.3	33.3	66.6	66.6	66.6	66.6	100	100	100	100
<i>Enterobacter aerogens</i>	0	0	0	66.6	50	50	50	50	66.6	33.3	33.3	66.6	66.6	100	100	100
<i>Pseudomonas aeruginosa</i>	60	0	0	100	0	0	50	50	100	100	100	100	0	100	100	100
<i>Escherichia coli</i>	0	0	0	100	0	0	100	100	100	100	100	100	0	100	100	100
<i>Citrobacter freundii</i>	0	0	0	100	0	0	100	0	100	100	0	100	100	100	100	1200

MRP-Meropenem, AMC-Amoxicillin, A/S-Ampicillin sulbactam, PIT-Piperacillin Tazobactam, CTX-Cefoxitin, CFS-Cefoperazone sulbactam, CPM-Cefepime, AK- Amikacin, GN- Gentamicin CIP- Ciprofloxacin, LE- Levofloxacin, TE- Tetracycline, IMP- Imipenem, CL- Colistin, PB- Polymyxin-B

Fig.2 Bar diagram Showing isolates from VAP cases

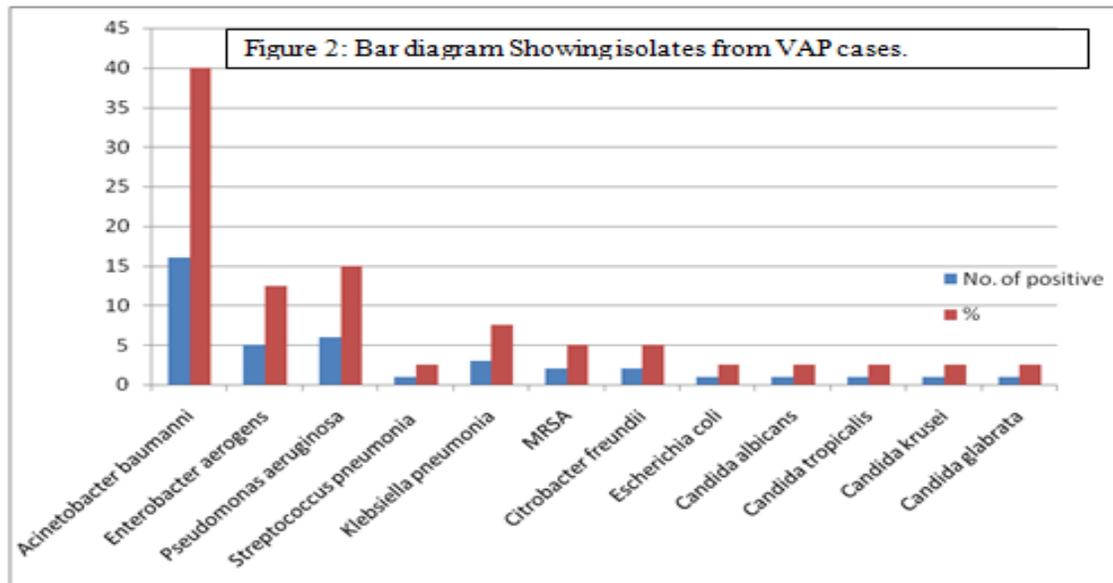
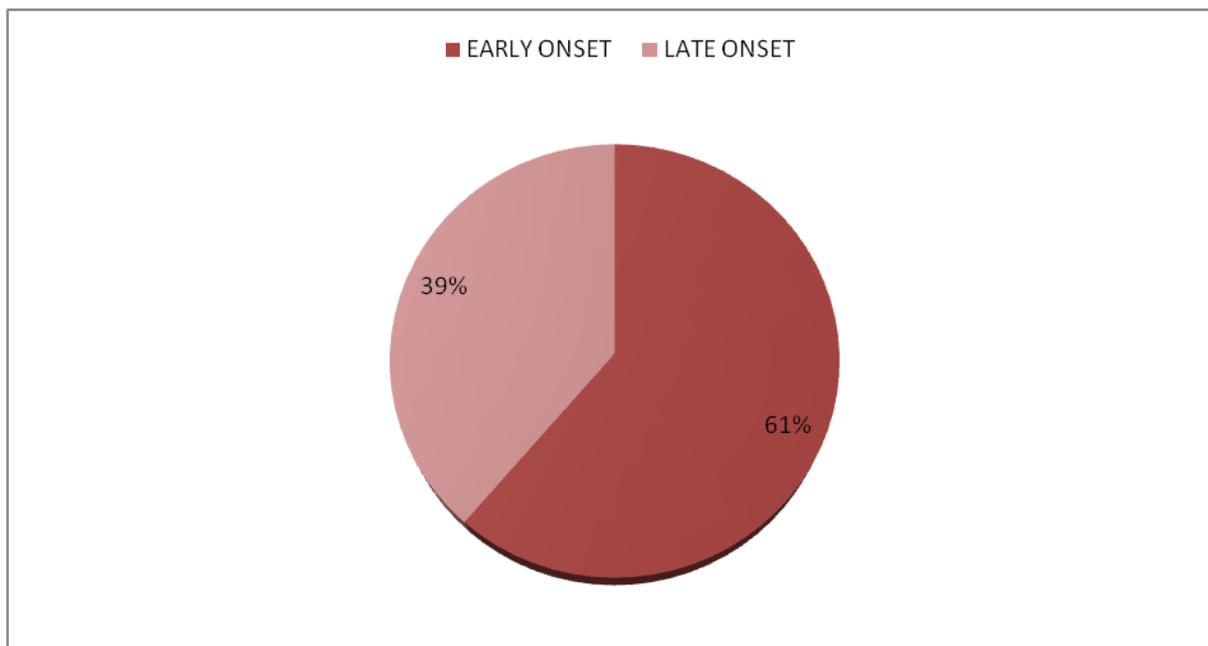


Fig.3 Distribution of early and late onset ICU



While Rajash karan *et al.*, who reported a very high incidence rate of 73%⁽¹⁷⁾. 18 The difference in the observation by this study might be due to the variation in the duration of hospitalization prior to ventilation, prior use of antibiotics, and type of invasive procedures. Our study showed that patients in the age group of 61-75 years were more prone to VAP as the number of patients exposed to mechanical ventilation (>48hours) were also more in this age group and this was found in accordance with Jakbrittu *et al.*,⁽¹⁸⁾ The incidence of VAP was more in males (64.51%) compared to females (35.48%) which was similar to studies conducted by Sharma *et al.*,⁽¹⁹⁾ Out of 31 VAP cases, 66 % were categorized under early-onset VAP and 34 % under late-onset VAP which was in concordance with studies conducted by Dey *et al.*,⁽²⁰⁾ and Chastre *et al.*,⁽¹³⁾ *Acinetobacter baumannii* (40%) and *Pseudomonas aeruginosa* (15%) were the commonest isolates obtained in both early and late onset VAP cases, which were also reported as the commonest isolates by other studies (Sharma *et al.*,⁽¹⁹⁾, Mukhopadhyay *et al.*,⁽²¹⁾ In this

study, *Acinetobacter baumannii* (40%), *Enterobacter aerogens* (12.5%) *Klebsiella pneumoniae* (7.5%) was most frequent in early-VAP cases whereas *Escherichia coli* (2.5%) and *Candida* spp in late-VAP case.

The recent study on nosocomial pneumonia in Asia including Korea also reported that *Acinetobacter* species, *P. aeruginosa*, *S. aureus*, and *K. pneumoniae* were the most common pathogens identified from both early and late nosocomial pneumonia. (Chung DR *et al.*,⁽²²⁾ this study positive cocci mainly *Staphylococcus aureus* (MRSA 5 %) and *Streptococcus pneumoniae* (2.5) the most frequently isolated organism in early onset VAP which is in contrast to our study.)⁽²³⁾ Restrepo *et al.*, Meropenem resistance was high in this study as 40% of the *Pseudomonas* and 37.5 % of *Acinetobacter* species showed multi-drug resistance (MDR), even to carbapenems, which is in concordance with other studies.^(24,20,25) Whereas certain studies reported a lower incidence of meropenem resistance.⁽²⁶⁾ 5% of *Staphylococcus aureus* strains were MRSA. Among the 2 MRSA

strains, 1 was isolated from patients with late onset VAP. The incidence of MRSA in our study correlates well with studies done by Gupta *et al.*,⁽²⁷⁾

The overall picture suggests that number of drug-resistant strains of various organisms is rising and is an important cause of VAP in our setting.

Therefore, this study suggests that most cases of VAP in our setting are those of late-onset VAP and a majority of these are caused by highly resistant strains and also the frequency of specific MDR pathogens causing VAP may vary by hospital, patient population, and exposure to antibiotics, type of ICU patients and changes over time, emphasizing the need for timely local surveillance data.

Early and accurate diagnosis is necessary to treat the patients in ICU. Proper monitoring and strict implementation of infection control practices is very essential to reduce VAP in hospital setting.

Also the short term use of invasive devices and judicious use of antibiotics are important in preventing VAP caused by these MDR pathogens. As for any HAI infection simple measures like hand washing, use of personal protective equipments such as gloves, face mask, and shoe cover can prevent infection thereby avoiding unnecessary use of antibiotic and will lead to cost curtailment.

Further, it will be prudent enough if a proper antibiotic protocol is developed (keeping in consideration the prevalent microorganism and their sensitivity pattern) and followed rigidly. It will prevent the emergence of resistant strains, and many commonly used drugs can be prevented from falling into the resistant category. The clinicians and the microbiologist should work in tandem to bring about the necessary changes.

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