A Dissertation

on

AN OBSERVATIONAL STUDY ON INCIDENCE , ETIOLOGY , RISK FACTORS , OUTCOME AND PREVENTION OF VENTILATOR ASSOCIATED PNEUMONIA IN IMCU PATIENTS ADMITTED IN GMKMCH , SALEM

Submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY CHENNAI

in partial fulfillment of the regulations for the award of the degree of

M.D BRANCH – I GENERAL MEDICINE

GOVT.MOHAN KUMARAMANGALAM MEDICAL COLLEGE & HOSPITAL, SALEM, TAMILNADU

MAY 2022

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled " **AN OBSERVATIONAL STUDY ON INCIDENCE , ETIOLOGY, RISK FACTORS , OUTCOME AND PREVENTION OF VENTILATOR ASSOCIATED PNEUMONIA IN IMCU PATIENTS ADMITTED IN GMKMCH , SALEM** " is a bonafide and genuine work carried out by me from November 2019 and november 2020 under the guidance and supervision of Professor **Dr. S.SURESH KANNA M.D.,** Professor and Head of the Department, Department of GENERAL MEDICINE, Government Mohan Kumaramangalam Medical College Hospital, Salem, Tamil Nadu, India.

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfilment of the requirement for the award of MD Degree in General Medicine(Branch I).

Date :

Place : SALEM

Signature of the Candidate

Dr. J. DIVYA

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Date :

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Signature of the Guide

Prof **DR. S. SURESH KANNA M.D**

Department of General Medicine Government

Mohan Kumaramangalam Medical College

Hospital, Salem

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Department of General Medicine Government

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Date :

Place : SALEM

Signature of the DEAN

Prof DR.VALLI SATHYAMOORTHY M.D, DA

THE DEAN

Government Mohan Kumaramangalam

Medical College Hospital, Salem

GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL

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GOVERNMENT MOHANKUMARAMANGALAM
MEDICAL COLLEGE & HOSPITAL
SALEM, TAMILNADU
College: Phone No.0427-2383313 Fax No:0427-2383193
E-Mail ID: deangmkmcslm@gmail.com
Hospital: Phone No: 0427 - 2210674, 2210757 Fax : 0427 - 2210876
E-Mail ID: msgmkmchsalem@gmail.com

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Guide/Principal Investigator	Dr.S.SURESH KANNA,MD, (Gem.Med.) Professor & HOD, DEPT. OF GENERAL MEDICINE GMKMCH, SALEM.
Primary Investigator	Dr.J.DIVYA,MD.,(GENERAL MEDICINE) 1st Year PG MD (GM) GMKMC&H, SALEM-01.
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Govt. Mohan Kumaramangalam
Medical College,
SALEM - 636 030.

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ABBREVIATIONS

VAP	-	Ventilator Associated Pneumonia
VAE	-	Ventilator Associated Events
IVAC	-	Infection Related Ventilator Associated Complications
BAL	-	Broncho Alveolar Lavage
CAUTI	-	Catheter Associated Urinary Tract Infections
CAP	-	Community Acquired Pneumonia
HAP	-	Hospital Acquired Pneumonia
HACP	-	Health Care Associated Pneumonia
COPD	-	Chronic Obstructive Pulmonary Disease
MDR	-	Multi Drug Resistant
ETT	-	Endotracheal Tube
MRSA	-	Methicillin Resistant Staphylococcus aureus
UTI	-	Urinary Tract Infection
LRT	-	Lower Respiratory Tract
ATS	-	American Thoracic Society
IDSA	-	Infectious Diseases Society of America
CPIS	-	Clinical Pulmonary Infection Score
FiO2	-	Fraction of oxygen in inspired air
PaO2	-	Partial pressure of oxygen
ETA	-	Endotracheal Aspirate

GNB	-	Gram negative bacilli
GPC	-	Gram positive cocci
CDC	-	Centre for Disease Control
GBS	-	Gullian Barre Syndrome
RHD	-	Rheumatic Heart Disease
MS	-	Mitral Stenosis
SLE	-	Systemic Lupus Erythematosus
DCLD	-	Decompensated Liver Disease
CFU	-	Colony forming unit
ICU	-	Intensive care unit
IMCU	-	Intensive medical care unit
GMKMCH	-	Government Mohan Kumaramangalam Medical College and Hospital
UZ	-	upper zone
LZ	-	lower zone

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ABSTRACT

BACKGROUND:

Ventilator associated pneumonia poses a major threat to patients admitted in intensive care units and receiving mechanical ventilation. 86% of nosocomial pneumonias are associated with mechanical ventilation and are termed ventilator associated pneumonia. Endotracheal intubation is the most important risk factor for developing VAP. Critically ill patients who are intubated for more than 24 hrs were found to be at 6 to 21 times higher risk of developing VAP , compared to non intubated patients.

AIMS AND OBJECTIVES:

- To study the incidence of VAP in IMCU of GMKMCH , Salem.
- To study the pathogens involved in the causation of VAP.
- To identify the percentage of early onset and late onset VAP.
- To identify the various risk factors involved in the development of VAP.
- To ascertain the significance of clinical pulmonary infection score CPIS in the diagnosis of VAP.
- To identify the association between ICU mortality and VAP

METHODOLOGY:

This is an observational study conducted in medical IMCU of GMKMCH, Salem for a period of one year from November 2019 to November 2020. Patients above the age of 18 yrs intubated in our institute and on mechanical ventilation for more than 48 hrs with normal chest X ray during

admission were included in the study. Patients intubated elsewhere , with abnormal chest X ray during admission and with pneumonia during admission or who develop pneumonia less than 48 hrs of intubation were excluded from study. Patient details , thorough history , relevant investigations were collected as per profoma. ETA was sent for all patients included in our study. The microbiological reports , duration of ventilation and the final outcome of the patient were noted. The variables plotted in Microsoft excel and analysed with relevant statistical methods.

RESULTS:

50 patients who satisfied the inclusion criteria were enrolled for the study. The incidence of VAP was observed to be 18% ie 9 patients developed VAP among the 50 patients enrolled for our study. The prevalence of early VAP was found to be 33.3% (3) compared to that of late VAP which was 66.7% (6). The age and gender distribution did not have statistical significance with the development of VAP. Emergency intubation(66.7%) had a significance in the development of VAP. 88.9% were gram negative organisms. Acinetobacter and pseudomonas each were observed to be 33.3% followed by klebsiella which was 22.2 % and CONS 11.1%. CPIS score of more than 6 had a statistical significance with the development of VAP ($P < 0.001$). The mean duration of ventilation was found to be 16 days in case of patients with VAP and 5 days in case of patients in NON VAP group. In our study there is no significant correlation between primary diagnosis and the development of

VAP.(P0.636).The mortality rate in our study was observed to be 33% in case of VAP and 41.5% among NON VAP group.

CONCLUSION:

VAP continues to be a commonly encountered problem amongst the critically ill patients admitted to the intensive care unit. It poses a great diagnostic and therapeutic challenge to the treating physician. A complete knowledge about the

Etiology , pathogenesis, risk factors and organisms involved is essential to combat VAP effectively. The diagnostic challenge of VAP has multiple implications for therapy. An exclusive microbiological profile and antibiogram is a must for every ICU. A complete knowledge about VAP would help in devicing effective preventive measures like antibiotic coated ETT , specialized ETT with programmed suctioning of the subglottic secretions.Antibiotic stewardship programs involving pharmacists , physicians and other health care personals would optimize antibiotic selection, dose and duration to increase efficacy intargeting causative organisms and thus would offer best clinical outcome.

KEYWORDS:

Ventilator associated pneumonia, Endotracheal tube , Microbiologiocal profile, ventilator bundle.

INTRODUCTION

Health care associated infections (HAI) result in significant economic and clinical burden on health care system. This burden is magnified by the increasing infection rates due to multi drug resistant (MDR) pathogens. Catheter associated urinary tract infections (CAUTI) are the most common nosocomial infection globally. Pneumonia is the second most common nosocomial infection and accounts for 15 to 20% of all nosocomial infections. Nosocomial pneumonia forms a very important etiology of hospital acquired infections and consists of the three distinct entities namely ventilator associated pneumonia (VAP) , health care associated pneumonia (HACP) and hospital acquired pneumonia (HAP) . Nosocomial pneumonia results in excessive health care utilization and also leads to greater mortality.

Nosocomial pneumonia refers to any pneumonia contracted by a patient admitted in a hospital at least 48 to 72 hours after admission to hospital and when it was not in incubation at the time of hospitalization. 86% of nosocomial pneumonias are associated with mechanical ventilation through endotracheal tube and are thus termed as ventilator associated pneumonia. VAP prevalence Varies between 6 to 52 cases per 100 patients, depending on the population under study.

It was observed that on any given day in the ICU usually an average of 10% of patients might have ventilator associated pneumonia. The frequency of diagnosis changes with the duration of mechanical ventilation. The highest hazard ratio is in the first 5 days and it is found to plateau after two weeks. Ventilator associated pneumonia is found to occur at rates of 10 to 35 cases/ 1000 ventilator days. The

approximate prevalence rate among patients who remain in ventilator for as long as 30 days is as high as 70%. Surprisingly once a ventilated Patient was transferred to a chronic care center or home the incidence of pneumonia drops significantly, especially in the absence of other risk factors for pneumonia.

Ventilator associated pneumonia arising 48 to 96 hours after tracheal intubation is usually called Early onset ventilator associated pneumonia and the one that occurs after this period is known as Late onset ventilator associated pneumonia. Early onset VAP has a better prognosis and more likely to be caused by aspiration of antibiotic sensitive bacteria colonizing the oropharynx. However late onset VAP maybe caused by multi drug resistant (MDR) pathogens and it is associated with greater morbidity and mortality.

Endotracheal intubation is an identified single major risk factor for developing ventilator associated pneumonia. The other factors being decreased level of consciousness, gastric distention, presence of ryles tube, trauma, COPD. Aspiration of oral or gastric fluids is recognized to be an important factor for the development of ventilator associated pneumonia. Pulmonary aspiration is increased by supine positioning and pooling of secretions above the cuff of the endotracheal tube.

The development of VAP is observed and ascertained to be directly proportional to the duration of mechanical ventilation. This in turn also prolongs hospital stay and also the hospitalization charges. It becomes extremely difficult and cumbersome to manage after the diagnosis of VAP. Various studies have also demonstrated failure rates of 49 to 62 % even after meticulous use of appropriate

standard antibiotic combinations. Hence it would be of paramount importance if each institute develops an ICU specific antibiogram and preventive strategies .

In this study the incidence of VAP, etiology ,common pathogens involved , risk factors and outcome of intubated patients admitted in IMCU of GMKMCH were analysed.

AIMS AND OBJECTIVES

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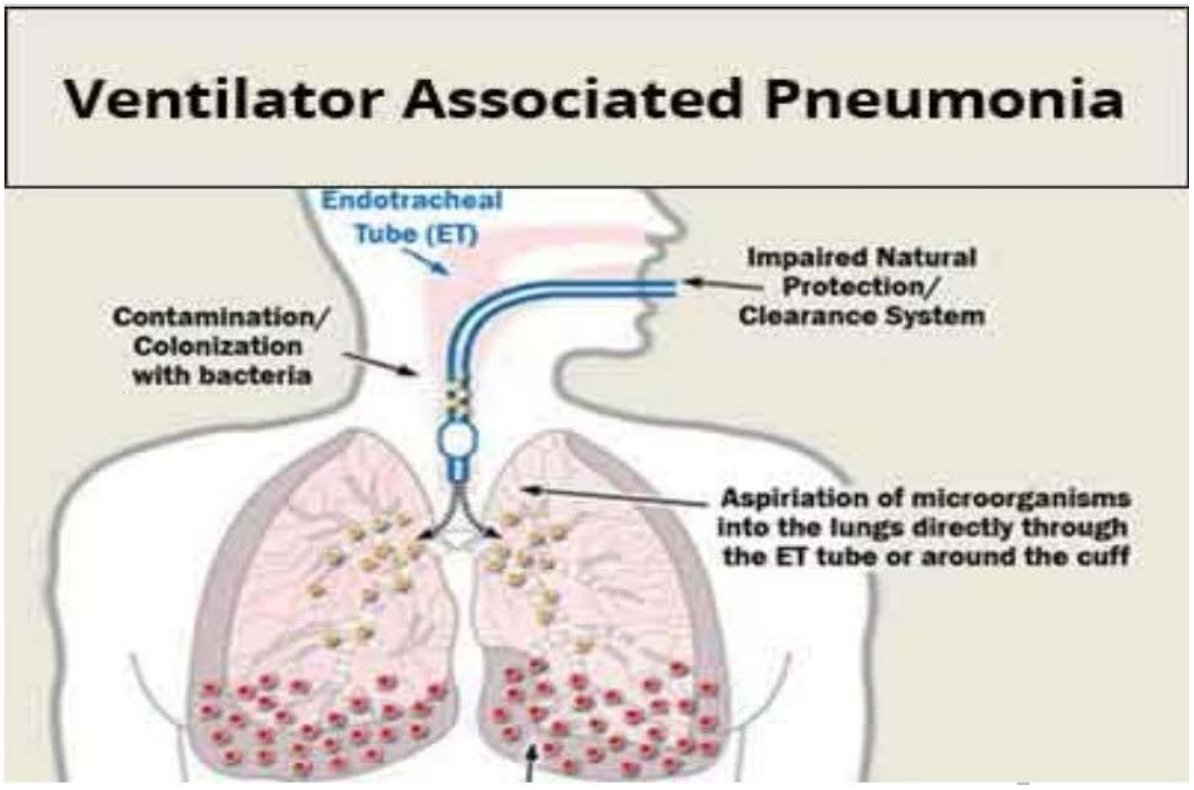
REVIEW OF LITERATURE

Pneumonia being the second most common nosocomial infection affecting the lung parenchyma is a leading cause of mortality and morbidity in the critically ill patients admitted in IMCU. Nosocomial pneumonia poses great diagnostic and therapeutic challenges to the treating clinician. Pneumonia accounts for approximately 24% of nosocomial infections and ventilator associated pneumonia (VAP) occurs in approximately 10% of patients who are on mechanical ventilators. These infections are observed to be responsible for 12–14 extra hospital days and increase the hospitalization charges by atleast 40% in extra costs per episode.



Most cases of VAP are caused due to aspiration of endogenous or hospital acquired oropharyngeal (and occasionally gastric) flora. VAP has been associated with more deaths than other infections at any other body site. The mortality rates due to VAP is affected greatly by other factors including comorbidities, inadequate antibiotic treatment, inadequate dose of antibiotic ,involvement of specific pathogens especially Pseudomonas aeruginosa or Acinetobacter and delay in the diagnosis of VAP. Follow up and accurate diagnosis of VAP have been challenging in hospitals because many patients, especially those in the ICU, have abnormal chest roentgenographs, fever, and leukocytosis potentially attributable to multiple other causes.

This diagnostic uncertainty and challenges has led to questions about the reliability of data and a renaming from VAP to ventilator-associated events (VAEs), for which worsening physiologic parameters, such as oxygenation, are the key factors. VAEs occur in as many as 5–10% of patients on mechanical ventilators. Risk factors for VAP include those events that cause colonization by potential pathogens (ie., previous antimicrobial therapy, contaminated ventilator circuits or equipments and decreased gastric acidity); those that potentiate aspiration of oropharyngeal contents into the lower respiratory tract (ie, intubation, decreased levels of consciousness, or presence of a ryles tube); and those that decrease host defense mechanisms in the lung and permit overgrowth of aspirated pathogens (ie, chronic obstructive pulmonary disease or upper gastrointestinal surgery).

Control measures for VAP are aimed at

- frequent testing of fitness for extubation , which can also shorten ICU stay
- taking extra caution in avoiding risk factors in patient care (ie., reducing aspiration-prone supine positioning); and
- strict aseptic care of respirator equipment.

Though the benefits of selective decontamination of the oropharynx and gut using nonabsorbable antimicrobial agents which is a practice avoided in the United States due to concerns about antibiotic resistance has been controversial, a randomized multicenter Dutch trial showed lower ICU mortality rates among those patients on mechanical ventilation who were given oropharyngeal decontamination.

Among the other preventive measures that require further study are providing channels for subglottic drainage of secretions in endotracheal tubes, which could reduce infection risks during short-term postoperative use and noninvasive mechanical ventilation whenever possible and feasible. It is also to be noted that just by reducing the rate of VAP alone donot warrant the reduction of overall ICU mortality. This suggests the inadequacies of surveillance and indicates that this infection at times is a marker for patients with a heightened risk of death.

Several important considerations regarding diagnosis and treatment are worth emphasizing.

- First,the currently used clinical criteria for diagnosis (ie., fever, leukocytosis, development of purulent secretions, new or change in radiographic infiltrates,

and change in oxygen requirement or ventilator settings) have high sensitivity but considerably low specificity. These criteria have been useful in selecting patients for either bronchoscopic or nonbronchoscopic procedures that yield with exclusive lower respiratory tract samples without upper-tract contamination. The quantitative cultures of such specimens collected have diagnostic sensitivities in the range of 80%.

- Second, early-onset VAP , which usually manifests within the first 4 days of hospitalization, is most likely to be caused by community-acquired pathogens like Streptococcus pneumoniae and Haemophilus species. Late-onset VAP are most commonly due to S. aureus, Pseudomonas aeruginosa, Enterobacter species, Klebsiella pneumoniae, and Acinetobacter.
- Third, one multicenter study suggests that 8 days can be considered as an appropriate duration of therapy for nosocomial VAP which lessen the emergence of resistant pathogens.
- Fourth and finally in febrile patients (especially those who have tubes inserted through the nose), occult bacterial sinusitis and otitis media should be considered without fail.

PATHOGENESIS

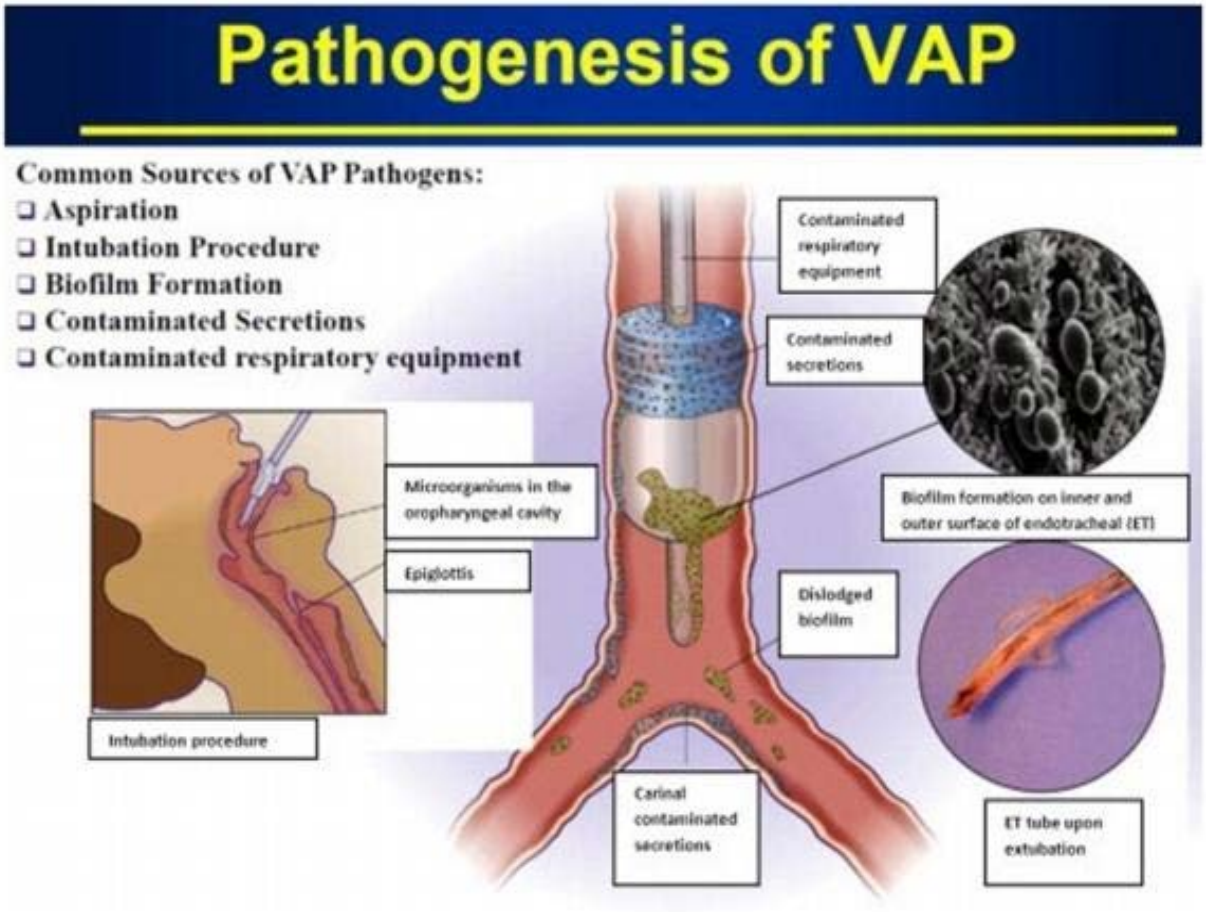
Pneumonia is a disease of the lung parenchyma due to the proliferation of microbial pathogens at the alveolar level and results due to the host's response to such pathogenic organisms. Microorganisms access the lower respiratory tract in several ways, aspiration from oropharynx being the most common cause. A Small-volume

aspiration occurs more frequently during sleep (especially in the elderly) and also in patients with decreased levels of consciousness. Sometimes, pneumonia occurs via hematogenous spread (ie., from tricuspid endocarditis) or by the direct extension from either infected pleural space or mediastinal space. The mechanical factors like the presence of hairs and turbinates of the nose capture large sized inhaled particles before they could manage to reach the lower respiratory tract and hence play a primary and major factor in host defence. The branching architecture of the tracheobronchial tree helps to trap microbes on the airway lining, where the potential pathogen is either cleared by mucociliary clearance or killed by local antibacterial factors. The gag and cough reflexes protect from aspiration. In addition, the normal flora which adheres to mucosal cells of the oropharynx, prevents pathogenic bacteria from binding and thus decreases the risk of pneumonia.Resident alveolar macrophages are extremely efficient at clearing and killing pathogens which overcome these barriers.

Macrophages assisted by proteins produced by the alveolar epithelial cells (ie., surfactant proteins A and D) and those that have intrinsic opsonizing properties or antibacterial or antiviral activity, engulf the pathogens and even if they are not killed will be eliminated either by the mucociliary elevator or by the lymphatics and no longer become an infectious challenge. When the capacity of the alveolar macrophages to ingest or kill the microorganisms is exceeded, then clinical pneumonia manifests. Thus it is the alveolar macrophages which initiate the inflammatory response inorder to boost the lower respiratory tract defenses.It is the host inflammatory response, rather than the proliferation of microorganisms, which

trigger the clinical syndrome of pneumonia. The release of various inflammatory mediators such as interleukin 1 and tumor necrosis factor result in fever. Chemokines like interleukin 8 and granulocyte colony stimulating factor stimulate the release of neutrophils which produce both peripheral leukocytosis and increased purulent secretions. Inflammatory mediators released by macrophages and the recruited neutrophils leak into the alveoli which is similar to acute respiratory distress syndrome, however in case of pneumonia this leak is localized (at least initially). Even the erythrocytes can cross the alveolar capillary membrane, thus causing hemoptysis .

This capillary leak results in a radiographic infiltrate and rales detectable on auscultation, and hypoxemia resulting from alveolar filling. In some cases bacterial pathogens appear to interfere with the hypoxemic vasoconstriction that normally occurs in fluid filled alveoli and this interference can result in severe hypoxemia. Decreased compliance as a result of capillary leak, hypoxemia, increased respiratory drive, increased secretions, and infection associated bronchospasm lead to dyspnea. If severe, these changes in lung mechanics secondary to reductions in lung volume and compliance and the intrapulmonary shunting of blood may result in respiratory failure and death.



STAGES OF PNEUMONIA

PHASE 1- EDEMA

Presence of a proteinaceous exudate and bacteria in the alveoli.

PHASE 2 – RED HEPATIZATION PHASE

Presence of erythrocytes in the cellular intra-alveolar exudate

PHASE 3 – GRAY HEPATIZATION

No new erythrocytes extravasate into the alveoli and those already present have been lysed and degraded. Neutrophil is the predominant cell, fibrin deposition is abundant, and bacterias have disappeared.

PHASE 4 – RESOLUTION

The macrophages reappear as the dominant cell type in the alveolar space, and the debris of neutrophils, bacteria, and fibrin are cleared.

In VAP, respiratory bronchiolitis may precede the development of a radiologically evident infiltrate. Microaspiration in nosocomial pneumonia results in bronchopneumonia pattern.

The aspiration of microorganisms colonizing in the aerodigestive tract of a host with altered immunity is the most common cause for nosocomial pneumonia. Either direct inhalation or hematogenous dissemination of bacteria is considered to be less frequent route of infection. Aspiration occuring during sleep is present in a large number of normal people however the volume aspirated is small and more importantly the pathogenicity of the microorganisms is found to be diminished. Hospitalized patients especially unconscious patients are considered more likely to aspirate larger volumes of virulent bacteria with higher frequency due to decreased sensorium, difficulty in swallowing, and impaired gut motility.

The significant role of colonization of the upper respiratory and digestive tracts has been extensively evaluated. Previous studies have significantly demonstrated that 23% of patients admitted to a medical ICU colonized with GNB developed nosocomial respiratory infection compared to only 3% of patients who were noncolonized .Later, genomic DNA analysis also proved that strains of bacteria isolated from the lower respiratory tract (LRT) of patients with nosocomial pneumonia were identical to those strains colonizing the oropharynx and/or GI tract.

ICU patients are at higher risk for colonization due to the additional potential for contaminated respiratory equipment , hospital water systems, the transmission of bacterial organisms through healthcare workers, the spread of microorganisms through the respiratory droplets, and pharmacologically induced alterations in the gastric pH. The endotracheal tube serves as a significant reservoir for bacteria from the upper airway to leak around the cuff and thus gain entry into the LRT. Few manufacturers have developed novel endotracheal tubes with have antimicrobial properties that prevent biofilm formation. Humidifiers and nebulizers used for intubated patients can be infected allowing for transfer of microorganisms between patients by respiratory personnel.

Critically ill patients on mechanical ventilation are prone to develop stress ulcers and thus are treated with histamine type 2 (H2) antagonists and proton pump inhibitors for the prevention of stress ulcers. Additionally, patients also frequently require enteral nutrition. Though these strategies provide effective prophylaxis and nutrition, however alterations in gastric pH promotes growth of bacteria in a normally sterile environment thus potentially increasing the risk for nosocomial pneumonia.

The hyperinflammatory response that occurs in ICU patients and risk for sepsis can cause multisystem organ failure and death. Improvements in the management of sepsis have decreased rates of early death due to sepsis but patients now often succumb to nosocomial pneumonias. Recent evidence shows that a subset of patients who survive the cytokine storm associated with the proinflammatory response of sepsis tend to develop an immunodeficient state which promotes viral reactivation and nosocomial infection especially pneumonia. Immunomodulator therapies are currently

being investigated to overcome this. Malnutrition, contaminated equipments, cross infection from other patients are the other significant observed additional risk factors. The most obvious and important risk factor for the development of VAP is the presence of endotracheal tube that tend to bypass the normal mechanical factors preventing aspiration. Although the endotracheal tube prevents the obvious large volume aspiration however microaspirations due to the secretions pooling around the cuff becomes inevitable. The endotracheal tube and the arising need for frequent suctioning finally damages the tracheal mucosa thus promoting its colonization by pathogens. These pathogens usually form a glycocalyx biofilm over the endotracheal tube's surface which is found to be more resistant to both antibiotics and also the host defense mechanisms.

In the critically ill IMCU patients the normal flora in oropharynx is mostly replaced by pathogenic microbes and almost all the intubated patients experience microaspiration and are found to be transiently colonized with these pathogens.But it is observed that not all the patients intubated develop VAP but only one third of colonized patients ultimately develop VAP, if the protective barriers are overcome or if the pathogens are considerably small enough to be inhaled to reach the alveolar levels, where they are supposed to be efficiently cleared and killed by the resident alveolar macrophages but manage to evade all these protective mechanisms.

PATHOGENIC MECHANISMS AND THEIR CORRESPONDING PREVENTIVE STRATEGIES

PATHOLOGY	PREVENTION
Colonization of oropharynx by pathogenic bacteria and elimination of normal flora	Avoid prolonged antibiotic course
Oropharyngeal aspiration – large volume during intubation	A short course of prophylactic antibiotics for comatose icu patients
Gastroesophageal reflux	Avoid high gastric residuals, use of prokinetic agents, postpyloric enteral feeding
Bacterial overgrowth of stomach	Selective decontamination of the digestive tract with nonabsorbable antibiotics
Cross infection from other patients	Hand washing and intensive infection control education of all health care personals, isolation and proper cleaning of all reusable equipments
Large volume aspiration	Rapid sequence intubation, decompression of stomach with ryles tube
Risk due to ET tube	Prefer noninvasive ventilation
Prolonged duration of ventilation	Meticulous weaning protocols and sleep vacations
Abnormal swallowing function	Early percutaneous tracheostomy
Secretions pooled above ET tube	Head end elevation, avoid reintubation, minimize seation and patient transport, frequent sterile suctioning
Altered lower respiratory host defences	Appropriate management of the underlying condition

MICROBIOLOGY

Etiologic agents responsible for nosocomial pneumonia depend on various factors including the underlying disease, geography, ICU population, duration of mechanical ventilation, previous antibiotic therapy, and finally the method used to obtain sample for respiratory cultures. Historically, aerobic gram-negative bacilli (GNB) form the most prevalent pathogens causing nosocomial pneumonia. Enterobacteriaceae are an important group of bacteria since the importance of aerodigestive colonization in the pathogenesis of VAP. Microorganisms in this group include Klebsiella, Escherichia coli, Enterobacter, Citrobacter, Proteus and the Serratia species. Other significant GNB include Pseudomonas, Acinetobacter, and Stenotrophmonas species. These bacteria are essentially ubiquitous in the environment and have minimal nutritional requirements enabling them suitable for the colonization of hospitalized patients.

EARLY ONSET VAP

The common organisms implicated in the development of early VAP are,

- Staph aureus (Staphylococcus aureus)
- H influenza (Hemophilus influenzae)
- Proteus species
- Serratia marcescens
- Strep. Pneumoniae (Streptococcus pneumoniae)
- Klebsiella pneumoniae.
- E.coli

LATE ONSET VAP

- Pseudomonas aeruginosa
- MRSA
- Acinetobacter species
- Enterobacter

Pathogens usually sensitive to antibiotics were found to be responsible for early onset VAP whereas pathogens which were multi drug resistant were found to be responsible for late onset VAP which were also more difficult to treat bacterial organisms. VAP can also occur due to polymicrobial infections. It is also observed that the prevalence of MDR organisms in VAP varies between institutions and also within the same institution depending on the ward. Rates of VAP were highest in the burns, surgical, trauma, and neurological/neurosurgical ICUs which ranged from 2.5 to 6 cases per 1000 ventilator days. VAP was diagnosed less frequently in medical and medical/surgical ICUs a rate of 1 to 1.8 cases per 1000 ventilator days.

ICU Location	VAP Cases	Ventilator Days	Rate[a]
Burn	89	15,379	5.8
Medical	208	153,408	1.4
Medical/Surgical	307	167,857	1.8
Neurologic	71	14,837	4.8
Neurosurgical	165	53,966	3.1
Surgical	374	106,736	3.5
Cardiothoracic	218	132,307	1.6
Trauma	555	92,460	6.0

[a]Expressed as number of VAP cases per 1000 ventilator days.
Source: Data from Dudeck MA, Horan TC, Peterson KD, et al. National Healthcare Safety Network (NHSN) Report, data summary for 2010, device-associated module. Am J Infect Control. 2011;39(10):798–816.

The risk factors for MDR organisms are:

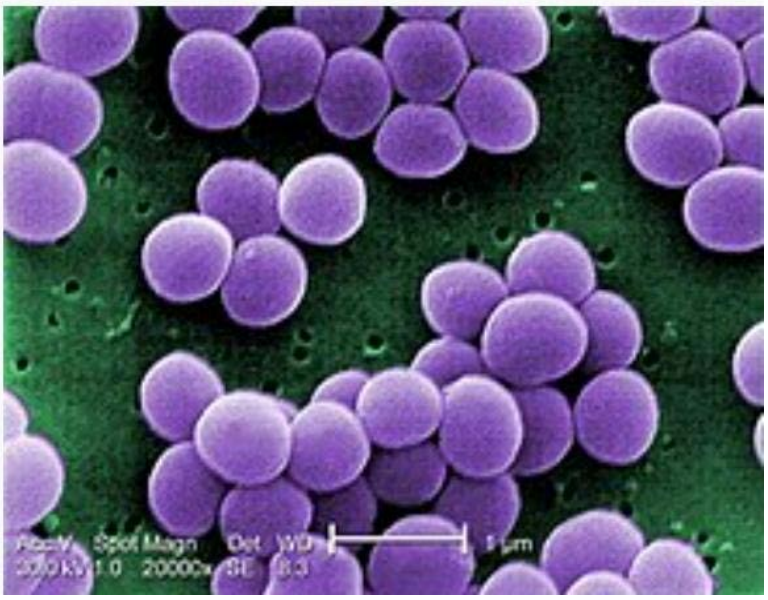
- Hospital inpatient admission for $\geq$ 2 days in the past 90 days.
- Patients put on chemotherapy or antibiotic therapy in the past 30 days.
- Patients who are on hemodialysis.

The usual commensals in oropharynx like the coagulase negative staphylococcus, Streptococcus viridans , Corynebacterium , and Neisseria species can also reach the lower airways in significant numbers causing VAP . Incidence of VAP due to fungal organisms and viral organisms are very low in immunocompetent host and their presence almost always indicate the presence of immunocompromised state.

The common pathogens involved in the causation of VAP, frequency and their possible modes of drug resistance are as follows:

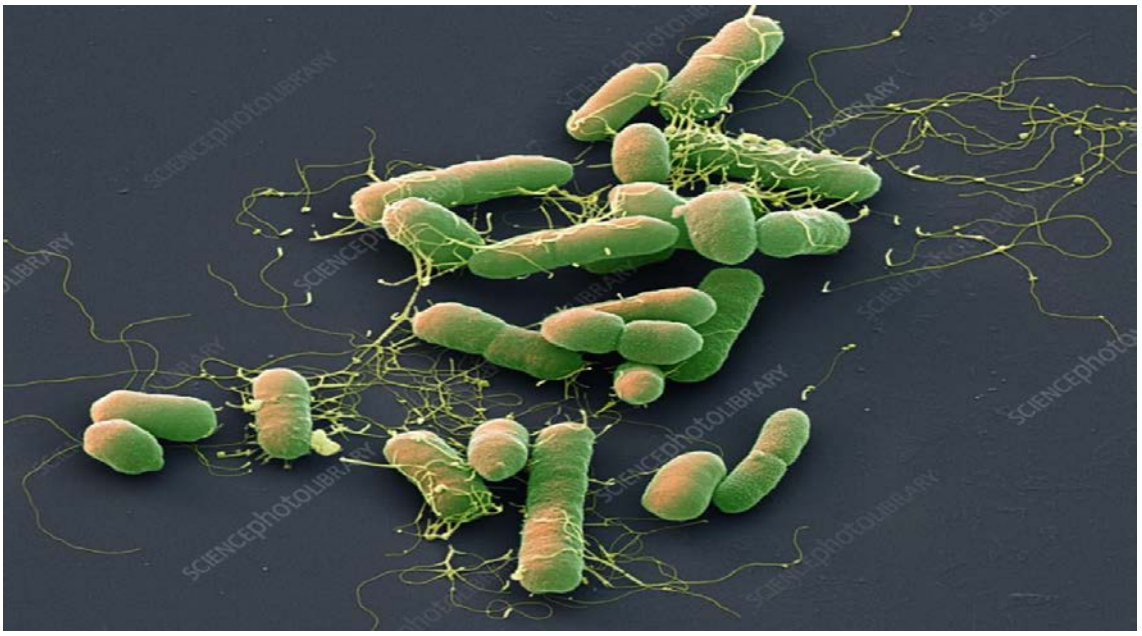
1) Pseudomonas (24.4%) - reduced expression of the outer membrane porin channel, acquire plasmid mediated metallo beta lactamases, increase regulation of efflux pumps.

2) Staphylococcus aureus (20.4% of which > 50% MRSA): It produces a pencillin binding protein (PBP) which has a decreased affinity for beta lactam antibiotics. PBP production is found to be encoded by the mecA gene.



3) Enterobacteriaceae (14% - includes Klebsiella spp; E.coli; Proteus spp., Enterobacter spp., Serratia spp., Citrobacter spp.) .There is plasmid mediated production of ESBL and plasmid mediated AmpC – type enzyme.

Klebsiella pneumoniae



Proteus mirabilis

4) Streptococcus species (12.1%)

5) Hemophilus species (9.8%)

6) Acinetobacter species (7.9%) – by production of metallo enzymes or carbapenamases.

7) Neisseria species (2.6%)

8) Stenotrophomonas maltophilia (1.7%)

9) Coagulase negative staphylococcus (1.4%)

10) Others (4.7% - includes Corynebacterium, Moraxella, Enterococcus, fungi).

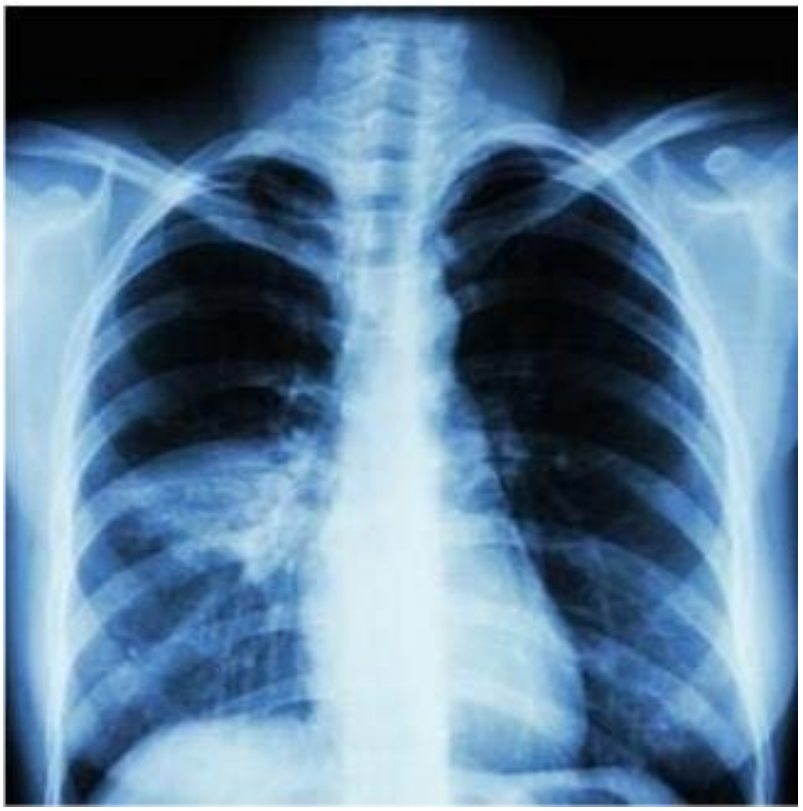
CLINICAL FEATURES

The latest definition requires a period of stability on a ventilator for atleast 48 hours for considering a ventilator associated event. Risk for developing VAP is highest during the first five days of mechanical ventilation and many standard studies have given the average duration between intubation and VAP development to be about 3.3 days. The occurrence of VAP has been linked to the use of mechanical ventilation over an extended period of time. After 5 to 10 days of ventilation, the risk is observed to decline to 2% per day, then to 1% per day after that.

The clinical features of VAP are

- pyrexia >38 degree celcius is one of the clinical symptoms of VAP.
- increased WBC count.
- Changes in sputum properties, as well as an increase in respiratory secretions.
- Physical examination reveals pulmonary consolidation
- Radiographic infiltration that is new or evolving
- Tachypnea and tachycardia are two symptoms of tachycardia.
- Decreased oxygenation
- Improvements in minute ventilation

- Culturing lower respiratory tract samples revealed the presence of a causal agent.
- The VAP rate is calculated as the number of VAPs divided by 1000 ventilator days.

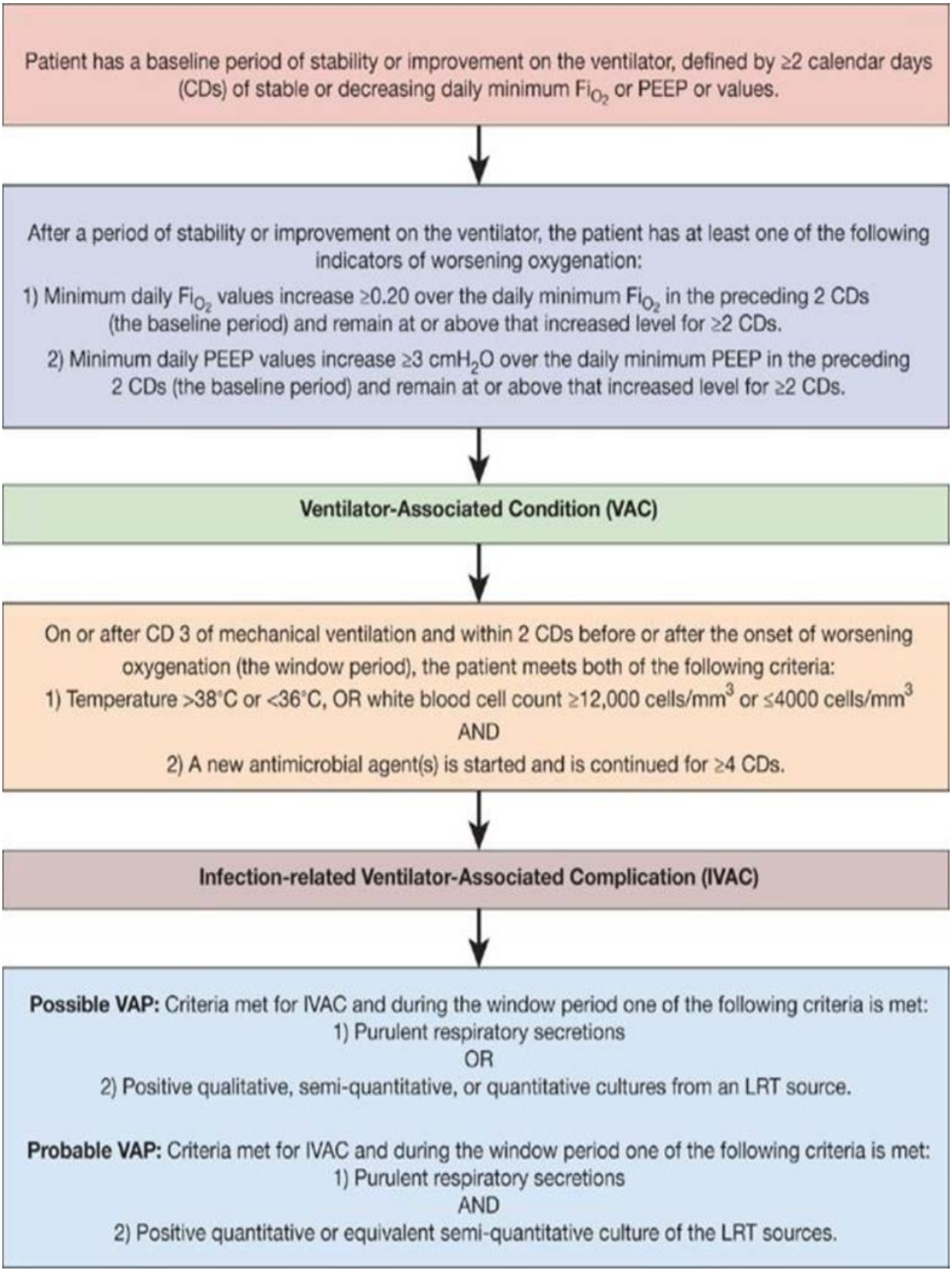


The three categories of ventilator associated events are:

i) Ventilator associated conditions (VAC)

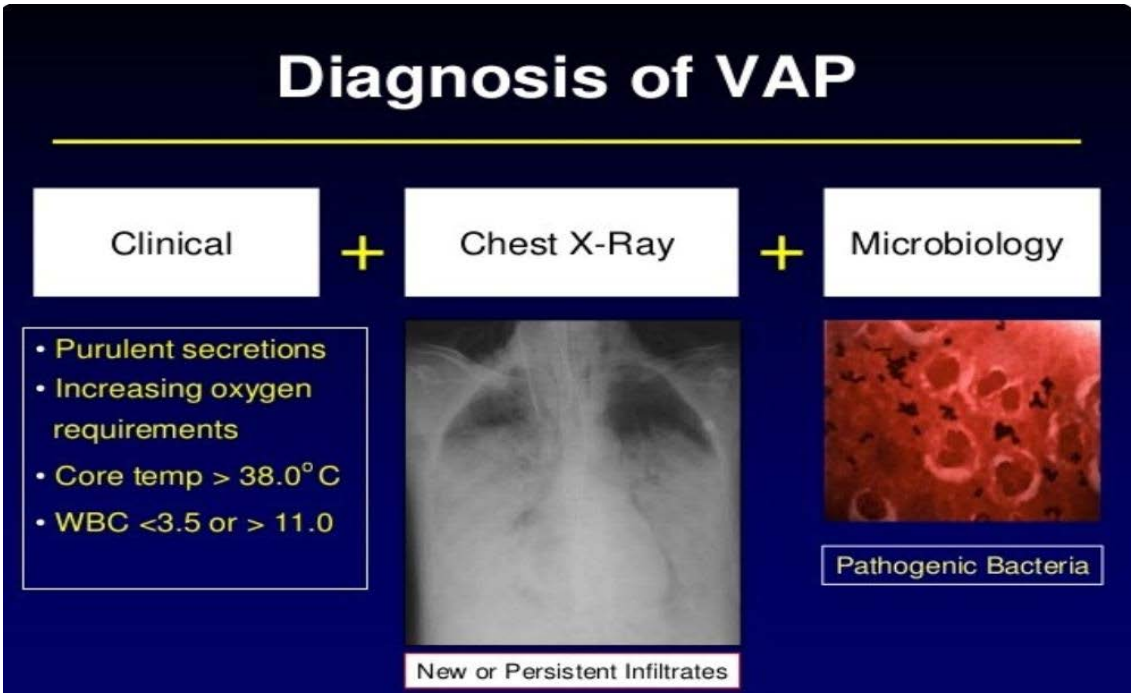
ii) Infection related ventilator associated complications (IVAC)

iii) Possible or probable VAP


Patient has a baseline period of stability or improvement on the ventilator, defined by ≥2 calendar days (CDs) of stable or decreasing daily minimum Fi_{O_2} or PEEP or values.
After a period of stability or improvement on the ventilator, the patient has at least one of the following indicators of worsening oxygenation:
1) Minimum daily Fi_{O_2} values increase ≥0.20 over the daily minimum Fi_{O_2} in the preceding 2 CDs (the baseline period) and remain at or above that increased level for ≥2 CDs.
2) Minimum daily PEEP values increase ≥3 cmH_2O over the daily minimum PEEP in the preceding 2 CDs (the baseline period) and remain at or above that increased level for ≥2 CDs.
Ventilator-Associated Condition (VAC)
On or after CD 3 of mechanical ventilation and within 2 CDs before or after the onset of worsening oxygenation (the window period), the patient meets both of the following criteria:
1) Temperature >38°C or <36°C, OR white blood cell count ≥12,000 cells/mm^3 or ≤4000 cells/mm^3
AND
2) A new antimicrobial agent(s) is started and is continued for ≥4 CDs.
Infection-related Ventilator-Associated Complication (IVAC)
Possible VAP: Criteria met for IVAC and during the window period one of the following criteria is met:
1) Purulent respiratory secretions
OR
2) Positive qualitative, semi-quantitative, or quantitative cultures from an LRT source.
Probable VAP: Criteria met for IVAC and during the window period one of the following criteria is met:
1) Purulent respiratory secretions
AND
2) Positive quantitative or equivalent semi-quantitative culture of the LRT sources.

DIAGNOSIS

There are no single set of criteria to reliably diagnose pneumonia in a ventilated patient. This inability to accurately identify such patients compromises the efforts to prevent and treat VAP successfully. This also results in inaccurate estimate of the impact of VAP on mortality rates.



Applying clinical criteria typical for CAP consistently results in overdiagnosis of VAP, because of the three common findings in at-risk patients:

(1) Frequent tracheal colonization with pathogenic bacterial organisms in patients with endotracheal tubes.

(2) Multiple other alternative causes of radiographic infiltrates in mechanically ventilated patients and

(3) The higher frequency of other sources of fever in critically ill patients.

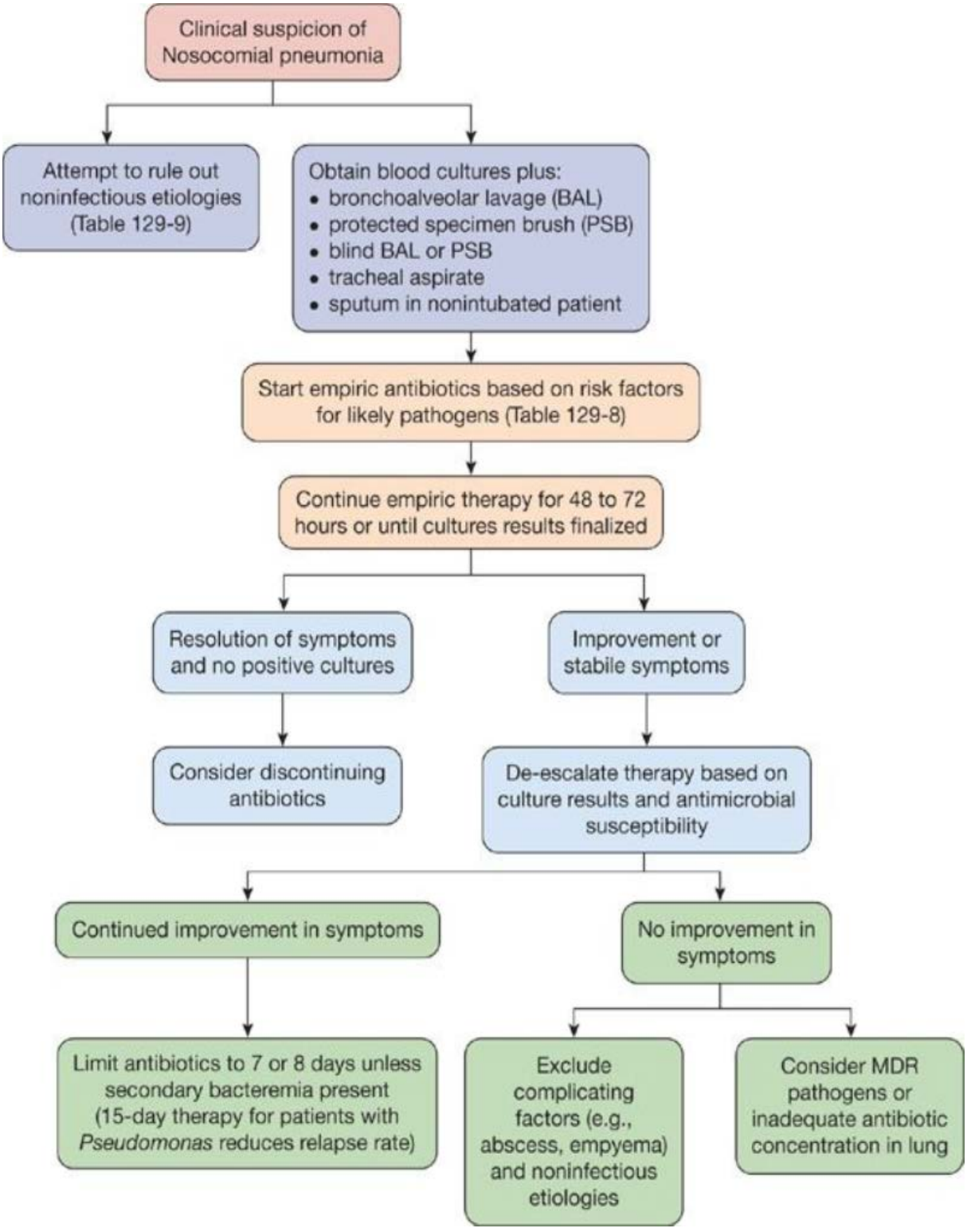
This diagnostic dilemma to diagnose VAP has led to debate and controversy. The major controversial question to be decided is whether a quantitative-culture approach used as a means of eliminating false-positive clinical diagnosis is superior to the clinical approach which is enhanced by principles learned from quantitative-culture studies.

Therefore a tailor made approach at each institution or preferably for each patient should balance the frequency of complex illnesses that are associated with

(1) higher frequency of alternative causes of the clinical manifestations,

(2) higher colonization rates

(3) more frequent prior antibiotic therapy against availability and expertise of invasive techniques to collect sample for quantitative cultures.



The clinical pulmonary infection score (CPIS) was developed which includes clinical, physiological, microbiological and radiographic evidence inorder to provide a numerical value to predict the presence or absence of VAP.

The CPIS score was originally developed by Pugin et al and others.

Table 1: Modified clinical-pulmonary infection score (clinical pulmonary infection score)

CPIS	0	1	2
Tracheal secretion	Rare	Abundant	Abundant and purulent
Chest X-ray infiltrate	No infiltrate	Diffuse	Localized
Temperature (°C)	36.5-38.4	38.5-38.9	≥39 or ≤36
Leucocytic count per mm^3	Within normal	4000-11000	>11,000\and\or band form 500
Hypoxic index PaO_2/FIO_2 mmHg	>240 or no ARDS	-	<240 and no evidence of ARDS
Microbiology	Negative	-	Positive

Any patient having score of 6 or more is considered having VAP. ARDS: Adult respiratory distress syndrome, PAO_2: Partial pressure of oxygen, CPIS: Clinical pulmonary infection score, VAP: Ventilator associated pneumonia[3-8]

The maximum score is 12. However , since the development of the infiltrate is uncertain at the time of diagnosis and tracheal aspirate cultures are also lacking, the initial maximum score is 8 to 10. The CPIS score ranges from 0 to 12.

A score of more than 6 indicates that VAP is present. CPIS scoringhas a se nsitivity of 93 percent and a specificity of 100 percent. Despite its widespreaduse, the validity of CPIS is still debated, as interobserver heterogeneity in the CPIS calculation impairs its application in clinical

Practice.According to the American Thoracic Society (ATS) and the Infectious Diseases Society of America (IDSA), lower respiratory samples for culture and micro biology are recommended.

The following are the numerous procedures for acquiring respiratory samples

- The simplest procedure is endotracheal aspiration.
- Bronchoalveolar lavage (BAL) this procedure necessitates the use of a broncho scopic scope.
- Minibronchoalveolar lavage (mini BAL) — a blind procedure performed Without the need for bronchoscopy
- Protected specimen brush.

The samples are quantitatively and qualitatively examined. There was also no difference in mortality between invasive and non-invasive groups, as well as quantitative versus qualitative cultures and antibiotic use.[18]After collecting the samples, they are sent for Gram staining, culture, and sensitivity testing. Gram stain is used to determine the type of organism and whether or not the material is purulent. Purulence is defined as the presence of more than 25 neutrophils and fewer than 10 squamous epithelial cells per low-power field[19].

The outcomes of the culture are given as semi-quantitative or quantitative numbers. Blood agar, Mac Conkey agar, and chocolate agar are all used to inoculate the samples. Semiquantitative values are deemed positive if agar growth is moderate (+++) or heavy (++++), whereas quantitative values are considered positive if > 105 cfu/ml. It takes a few days for the particular species of the organism involved and their antibiotic susceptibility to be disclosed, but it is crucial information.

DIFFERENTIAL DIAGNOSIS

- Atypical pulmonary edema
- Pulmonary contusion
- Hypersensitivity pneumonitis
- Pulmonary embolism
- Alveolar hemorrhage
- Acute respiratory distress syndrome.

TREATMENT

The antibiotic of choice is mostly determined by the length of mechanical ventilation. Early-onset VAP only requires limited-spectrum antibiotics, whereas late-onset VAP may necessitate the use of broad-spectrum antibiotics. The initial empirical therapy is guided by a periodically updated local antibiogram for each hospital and ICU based on local bacteriological patterns and susceptibilities.

De-escalation of the empirical antibiotic regimen is critical for preventing the formation of resistance, yet delaying antibiotic therapy may increase the risk of VAP-related mortality.[20] Early onset VAP treatment lasts 8 days, while late onset VAP treatment lasts much longer. The treatment time for VAP caused by MDR pathogens is usually around 14 days. [21] Antibiotic treatment should be halted after 8 days if the CPIS score falls during the first three days. An eight-day course is thought to be the most effective.

TABLE 121-8 Empirical Antibiotic Treatment of Hospital-Acquired and Ventilator-Associated Pneumonia

NO RISK FACTORS FOR RESISTANT GRAM-NEGATIVE PATHOGEN	RISK FACTORS FOR RESISTANT GRAM-NEGATIVE PATHOGEN[a] *(CHOOSE ONE FROM EACH COLUMN)*	
Piperacillin-tazobactam (4.5 g IV q6h[b])	Piperacillin-tazobactam (4.5 g IV q6h[b])	Amikacin (15–20 mg/kg IV q24h)
Cefepime (2 g IV q8h)	Cefepime (2 g IV q8h)	Gentamicin (5–7 mg/kg IV q24h)
Levofloxacin (750 mg IV q24h)	Ceftazidime (2 g IV q8h)	Tobramycin (5–7 mg/kg IV q24h)
	Imipenem (500 mg IV q6h[b])	Ciprofloxacin (400 mg IV q8h)
	Meropenem (1 g IV q8h)	Levofloxacin (750 mg IV q24h)
		Colistin (loading dose of 5 mg/kg IV followed by maintenance doses of 2.5 mg × [1.5 × CrCl + 30] IV q12h)
		Polymyxin B (2.5–3.0 mg/kg per day IV in 2 divided doses)
Risk Factors for MRSA[b] *(Add to above)*		
Linezolid (600 mg IV q12h) *or*		
Adjusted-dose vancomycin (trough level, 15–20 mg/dL)		

[a]Prior antibiotic therapy, prior hospitalization, local antibiogram. [b]Prior antibiotic therapy, prior hospitalization, known MRSA colonization, chronic hemodialysis, local documented MRSA pneumonia rate >10% (or local rate unknown).

Abbreviations: CrCl, creatinine clearance rate; MRSA, methicillin-resistant *Staphylococcus aureus*.

Initial Empiric Antibiotic treatment for early onset HAP (Table I)

Potential Pathogen	Recommended Regimen*
❖ Strep. Pneumoniae$ ❖ H. Influenza ❖ MSSA ❖ Antibiotic – sensitive enteric Gram –ve bacilli: ❖ E coli ❖ K. pneumoniae ❖ Enterobacter sp. ❖ Proteus sp. ❖ S. marcescens	❖ 3rd generation cephalosporins (ceftriaxone, cefotaxime) or ❖ Fluoroquinolones (moxifloxacin, levofloxacin) or ❖ ß-lactum/ß-lactamase inhibitor (amoxicillin/clavulanic acid; ampicillin/ sulbactum) or ❖ Carbepenms (ertapenem) or ❖ 3rd generation cephalosporins + macrolide or ❖ monobactum + clindamycin (for ß-lactum – allergic patients

*Antibiotic options should depend on local epedimiology of etiologic pathogens.
$ The frequency of macrolide-resistant S. pneum and MDR S pneum is increasing; levofloxacin or moxifloxacin ae preferred to ciprofloxacin and the role of other new quinolones has not been established.

Song JH and Asian HAP Working Group, Am J of Inf Control; vol 36; issue 4 May 2008; S83-S92

Initial Empiric Antibiotic treatment for late onset HAP (Table II)

Potential Pathogen	Recommended Regimen
Pathogens Listed in table I and MDR pathogens ❖ Pseudomonas aeruginosa ❖ K. pneumoniae (ESBL+)* ❖ Acinetobacter sp. * MRSA Legionella pneumophilia *	➢ Antipseudomonal cephalosporins (cefepime, ceftazidime) or Antipseudomonal carbepene (imipenem or meropenem) or ß-lactum/ß-lactamase inhibitor (piperacillin - tazobactum) +/- Fluoroquinolone (ciprofloxaccin or levofloxacin) or Aminoglycoside (amikacin, gentamicin or tobramycin) ➢ Cefoperazone / sulbactum + fluoroquinolones or aminoglycosides + ampicillin/ sulbactum (if sulbactum is not available or Fluoroquinolones(ciprofloxacin) + aminoglycoside + linezolid or glycopeptide (vancomycin or teicoplanin)$ + azithromycin of fluoroquinolone

*if an ESBL strain such as K. pneum or an Acinetobacter sp. Is suspected the a carpenem is a reliable choice. If L. pneumophilia is suspected then the combination antibiotic regimen should include a macrolide (eg. Azithromycin) or a fluoroquinolone (eg. Ciprofloxacin or levofloxacin) should be used rather than an aminoglycoside.
$ If MRSA risk factors are presenr or there is a high incidence locally.

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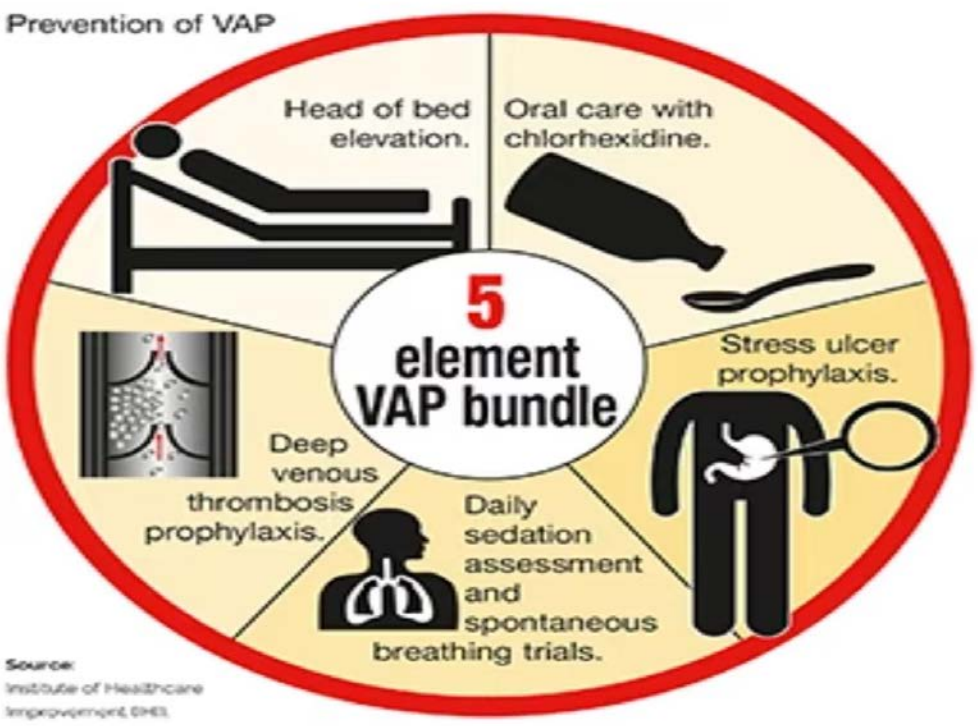
PREVENTION OF VAP

Prevention is usually preferable to cure, especially in the case of VAP, which is a preventable condition in which a meticulous approach minimises hospitalisation time, cost, morbidity, and death. VAP can be prevented using a variety of effective methods.



- **AVOID ET TUBE** :

Several studies have focused on the critical function of the endotracheal tube (ETT) in the aetiology of ventilator-associated pneumonia throughout the last several decades. Tracheal intubation suppresses the cough reflex, impairs mucocilliary clearance, damages the tracheal epithelial surface, permits germs from the upper to

enter the lower respiratory tract quickly, and allows biofilm to build on the ETT surface. The combination of these factors increases the risk of ventilator-associated pneumonia in mechanically ventilated patients. Controlling intracuff pressure, aspirating subglottic secretions, decontaminating the subglottic area, using antiseptic-impregnated ETTs, and eliminating or preventing ETT biofilm formation are just a few of the preventive techniques that have emerged as a result of this knowledge. Because the presence of an endotracheal tube is a considerable risk factor for the development of VAP, the most important preventive approach is to avoid or shorten endotracheal intubation if possible. When available, noninvasive ventilation is a good alternative to endotracheal intubation.

- **HEALTHCARE PROFESSIONALS :**

Nursing staff have a critical role in preventing bacterial colonisation of the oropharynx and gastrointestinal tract as the first line of defence. Before and after interaction with patients, proper hand cleaning with alcohol rub for 10 seconds should be conducted. Hand sterilisation should be done after and before attending to every patient, and separate sterile gloves should be used for each patient.

- **ORAL DECONTAMINATION** :

The quantity of microorganisms in a patient's oral cavity is reduced by proper oral hygiene and disinfection. Brushing the teeth and washing the oral cavity to remove dental plaque are examples of mechanical interventions. Suctioning also aids in the removal of plaque from the teeth. Antimicrobial drugs such as chlorhexidine oral rinse are used twice a day in pharmacological therapies. [(26)]Use of chlorhexidine

has been shown to reduce colonisation and the incidence of VAP in patients after heart surgery. [27] Except for patients undergoing heart surgery, there is no evidence-based oral care programme that has been shown to reduce the occurrence of VAP. VAP can also be prevented by using a solution comprising gentamycin, colistin, and vancomycin every 6 hours[28].

- **STRESS ULCER PROPHYLAXIS:**

Patients who have been on mechanical breathing for more than 48 hours are 16 times more likely to get gastro intestinal haemorrhage. [29] Stress ulcer prophylaxis, which raises gastric pH, is administered to patients on mechanical ventilation. In the alkaline gastric environment, pathogens grow, and bacterial colonisation of the stomach can lead to aspiration and colonisation of the respiratory system. [30]H2 receptor blockers, like as ranitidine, minimise the risk of bleeding while also lowering the risk of VAP and mortality.[31] Stress ulcer prophylaxis though does not play any pivotal role in the development of VAP it still prevents gastrointestinal bleeding.

- **FREQUENT SUCTIONING** :

Suction of the subglottic area with intermittent or continuous aspiration of subglottic secretions may lessen the risk of aspiration and VAP by removing contaminated oropharyngeal secretions accumulated above the ETT cuff. Specially designed ETTs with a separate dorsal lumen that enters into the subglottic space are required for aspiration of subglottic secretions (Hi-Lo Evac tube; Mallinckrodt, Athlon, Ireland). In order to liquefy secretions and prevent mucus blockages in endotracheal tubes, saline lavage has also been utilised. Saline lavage of endotracheal

tubes before suctioning, on the other hand, dislodges bacteria into lower airways, increasing the total risk of VAP. [34] It was also shown that saline instillation did not liquefy secretions, but rather lowered the quantity of oxygen reaching the lungs, resulting in increased blood pressure, heart rate, intracranial pressure, and the risk of VAP. [35] As a result, appropriate hydration of the patient, proper humidification of the ventilator circuit, and the administration of adequate nebulizer or mucolytic agents are essential methods for avoiding the need for saline lavage. It was also discovered that systemic antibiotic prophylaxis had no effect on VAP incidence.

The effectiveness of suction of subglottic secretions has been studied in five randomised controlled studies. In four trials, the incidence of VAP was found to be statistically significant lower. The time it took for VAP to develop was significantly delayed in all five trials. Only one study found that aspirating subglottic secretions reduced the incidence of trachea colonisation. Nonetheless, aspiration of subglottic secretions appears to have no influence on mortality, mechanical ventilation time, or intensive care unit or hospital stay.

- **FREQUENT CHANGE OF POSITION** :

Patients' positions are changed every 2 hours, which improves pulmonary drainage and thereby decreases the development of VAP. The use of customised beds capable of continuous lateral rotation would reduce pneumonia incidence but neither death or mechanical ventilation duration. As a result, these beds are not commonly utilised to prevent VAP [37]. Patients with poor oxygenation or poor wound healing can benefit from special beds that are cost-effective.

- **VENTILATOR CIRCUIT** :

The colonisation of the ventilator circuit is crucial to the progression of VAP. The incidence of VAP is not reduced by changing the ventilator circuit on a regular basis. [38]The Centers for Disease Control and Prevention recommends changing the ventilator circuit no more than once per 48 hours, and it has been shown that changing the ventilator circuit once a week does not increase the risk of VAP. [39]Changing the ventilator circuit simply when it becomes clearly filthy is adequate. The impact of heat and moisture exchangers on the definitive incidence of VAP as well as the type of humidity that is linked to enhanced VAP incidence is equivocal.

- **WEANING AND EARLY EXTUBATION** :

The most critical factor in the development of VAP is the presence of an endotracheal tube. As a result, patients should be monitored on a daily basis for the possibility of weaning and early extubation. T-piece trials, tapering intermittent mandatory ventilation, and providing pressure support ventilation can all be used to determine extubation readiness. [40]

- **HEAD END ELEVATION :**

Gastric reflux and aspiration into the airways are effectively prevented by positioning patients in a semirecumbent position with head end elevation of 30^{o} to 45^{o}. VAP is reduced by 34% with this simple procedure of elevating the head end of the bed by 30 degrees. [41]

- **SLEEP VACATION :**

Aspiration of stomach contents can be avoided by reducing the use of

narcotics. [42] It is not advisable to reduce the use of opioids and sedatives in an indiscriminate manner, as pain inhibits deep breathing and, as a result, oxygenation. The careful daily interruption of continuous sedative infusions effectively reduces mechanical ventilation by more than 2 days and consequently the ICU stay by 3.5 days. [43]

- **HALF STOMACH :**

As a method to prevent VAP, gastric overdistension should be avoided by monitoring gastric residual volumes and administering medicines that promote stomach motility. [44] Ryles tube feeding should be done with caution, and tiny, frequent feeds should be administered.

- **CUFF PRESURE :**

Because secretions pool above the cuffs of the endotracheal tubes, the patient's oropharynx should be thoroughly suctioned often while on endotracheal intubation. To avoid secretion leakage and aspiration, a sufficient cuff pressure must be maintained. Endotracheal tubes with ports for continuous suctioning lower the incidence of VAP by 50% and should have a pressure in the cuff of no less than 20cm of H_2O[45]. [46]

- **PREVENTING FORMATION OF BIOFILM :**

Another essential role of the ETT in VAP pathogenesis is that it acts as a reservoir for bacteria by providing a surface for them to attach to. To put it another way, it enables microorganisms to create a biofilm. A biofilm is defined as "a organised community of bacterial cells encased in a self-produced polymeric matrix

and attached to an inert or living surface," according to Costerton et al. A biofilm is a continuous source of infection that protects microorganisms against antibiotics by the accumulation of this protective glycocalyx. Thus, microbial resistance in biofilms appears to be based on multicellular tactics distinct from the already well-known plasmids, transposons, and mutations that impart inherent resistance to individual microorganisms. After intubation, biofilm builds quickly on the ETT and looks to be a substantial problem. Suction catheters may easily separate ETT biofilm aggregates, which are then distributed towards the lower respiratory tract by shear forces. Feldman et al. investigated ETT colonisation in mechanically ventilated patients and discovered that secretions lining the inside of the distal third of the tube produced a biofilm. In an observational research, Adair et al. looked into the association between ETT biofilm and VAP, finding that 70% of patients with VAP had microorganisms identified from both ETT biofilm and tracheal secretions.

Biofilm Formation Prevention

Three approaches can be used to prevent ETT biofilm formation:

- **SDD decontamination** : tobramycin , polymyxin , amphotericin B were found to eliminate only the colonization of gram negative organisms

- **use of specialised antiseptic impregnated ETT** : silver which is nontoxic is a highly effective antibacterial substance. A study by Rello et al confirms that silver coated ETT was well tolerated and associated with reduced bacterial burden in tracheal aspirates . This measure may reduce airway colonization by

bacterial pathogens associated with VAP, but more extensive studies are indeed needed inorder to determine if this decreases incidence of VAP.

- **synchronised mucus aspiration in the distal end of ETT :** Mucus slurper which is a modified ETT allows automatic aspiration of all secretions as it reaches the ETT. Animal studies have been found to be effective but similar clinical studies on mechanically ventilated patients are required to confirm these findings.

- **OUTCOME MANAGER :**

In order to give a holistic strategy to VAP prevention, an outcome manager can be hired. According to a study, using an exclusive outcome manager can reduce the length of mechanical ventilation, hospital stay, and mortality. [48]

- **EDUCATING THE NURSING STAFF:**

The impact of VAP on morbidity, mortality, length of stay in the hospital, and cost is enormous. As a result, educating all healthcare personnel involved is critical in the management of VAP. The rate of pneumonia, the duration of mechanical ventilation, and eventually the expense of the disease are reduced when support workers in the ICU get self-study education modules on the nursing care of patients on mechanical ventilators who are at risk of developing VAP. (49)

As a result, using a suitable VAP bundle method in the ICU can minimise VAP rates.

Measures for preventing VAP[50, 51, 52]

ICU focussed measures	Institution focussed measures
- Alcohol rub for hand washing - Discontinuation of invasive devices at the earliest. - Avoiding re-intubationrates. - Usage of oropharyngeal rather than nasopharyngeal feeding tubes. - Semi recumbent positioning of the patient (30-45^{o}) - Maintaining endotracheal cuff pressure at 20cm of H2O - Early initiation of tracheostomy. - Small bowel feeding ratherthan gastric feeding. - Use of probiotics.	- Measures for profiling of pathogens and creating an antibiogram. - Avoidance of using of unnecessary antibiotics. - Use of non-invasive positive pressure ventilation whenever feasible. - Use of antibiotic or silver coated endotracheal tube. - Oropharynx decontamination policies. - Selective digestive decontamination (SDD) - Sedation vacations - Early weaning and extubation protocols. - Removal of biofilm mechanically by mucus shaver.

COMPLICATIONS OF VAP : [53]

- Death is the most important complication of VAP.
- VAP prolongs mechanical ventilation and lengthens the time spent in the intensive care unit and in the hospital. This results in additional costs.

- Pseudomonas aeruginosa causes necrotising pneumonia, which results in substantial pulmonary bleeding.
- These necrotizing infections cause bronchiectasis and pulmonary scarring, which can lead to recurrent pneumonia episodes in the long run.
- Pneumonia causes a catabolic state in a patient who is already nutritionally deficient.
- Following ventilator-associated pneumonia, muscle loss and overall debilitation necessitate protracted rehabilitation, reliance on caregivers for functioning, and the need for nursing care.

FOLLOW UP IN VAP : [53]

- Clinical improvement usually occurs 48-72 hours after starting antibiotics.
- Until they are weaned off of mechanical ventilation, patients are subjected to daily serial chest radiographs.

PROGNOSIS : [53]

- It has been discovered that VAP and ICU mortality have a substantial relationship.
- The crude death rate is 50-70 percent, but attributable mortality is the most critical concern.
- Patients with VAP often have underlying co-morbid illnesses that, even if VAP did not occur, might lead to mortality.

- Previously, attributable mortality rates were higher than 25%, but they are now significantly lower.
- Variability in VAP mortality rates is linked to the patient's condition as well as the type of ICU.
- VAP in trauma patients is not related to attributable mortality becausethe patients were otherwise previously healthy.
- The most critical element in deciding the outcome of VAP is the causative pathogen. MDR pathogens are linked to a higher level of morality than non-MDR infection.
- Nosocomial pneumonia caused by Stenotrophomonas maltophilia indicates that the patient's immune system has been compromised, and death is nearly certain.

MATERIALS AND METHODS

STUDY DESIGN	:	Observational study
SAMPLE SIZE	:	50
STUDY PERIOD	:	November 2019 to November 2020
PLACE OF STUDY	:	Medical IMCU of General Medicine department in GMKMCH , Salem.
SELECTION OF CASES	:	Patients on mechanical ventilation for more than 48 hrs in IMCU.
ETHICAL COMMITTEE	:	Approval obtained.
CONFLICT OF INTEREST	:	None

METHODOLOGY :

This is an observational study conducted in the Intensive Medical Care unit of GMKMCH , Salem for a period of one year from November 2019 to November 2020. Patients admitted to the medical IMCU of GMKMCH above 18 years of age who were on mechanical ventilation following endotracheal intubation in our institute for more than 48 hours irrespective of the primary etiology were included in our study . Patients with normal chest x ray at admission and without any history of symptoms suggestive of any respiratory disease with recent onset were only included for the study. A total of 50 patients were included in the study. Modified Clinical Pulmonary Infection Score was used to aid clinically diagnose VAP. Detailed history of the patient including the name, age ,sex ,date of admission to IMCU , underlying risk factors , primary diagnosis during admission, treatment being administered ,number of

days on mechanical ventilation and the clinical outcome of each patient was noted . Patients who developed symptoms like fever , increased leukocytes , new infiltrates in chest xray , needed increase in ventilator settings were selected and their tracheal aspirate obtained through suction catheter inserted through endotracheal tube collected and sent for ETT culture sensitivity. Any lower respiratory tract infection that developed after 48 hours of continuous mechanical ventilation and was confirmed not to have been incubating before mechanical ventilation was considered to be as VAP. The diagnosis of VAP was based on the clinical and microbiological criteria. A clinical suspicion of VAP was made in patients who had modified CPIS score ≥ 6. The diagnosis was confirmed when significant growth of organism was obtained in the endotracheal tube culture sample obtained through the suction catheter. Endotracheal aspirate (ETA) sample was collected from all the patients included in the study admitted in the IMCU requiring mechanical ventilation for more than 48 hours. Patients who were already on mechanical ventilation before admission to the GMKMCH, IMCU and patients who had pneumonia or developed pneumonia less than 48 hours of admission were excluded. Grams stain preparation and culture of all endotracheal aspirate samples were done.

INCLUSION CRITERIA:

- Patients above the age of 18 yrs of age who were on mechanical ventilation for more than 48 hours irrespective of the primary etiology.
- Patients with normal CXR on admission
- Either sex

EXCLUSION CRITERIA:

- Patients intubated outside.
- Patients with pneumonia or who developed pneumonia less than 48 hrs of mechanical ventilation.
- Patients with abnormal chest x ray on admission.
- Decline of consent by guardian.

STATISTICAL ANALYSIS :

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram and pie diagram.

All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of >0.05 was considered as normal distribution.

Inferential statistics:

Quantitative outcome;

The association between VAP and no VAP and Duration of ventilation (days) was assessed by comparing the mean values. Independent sample t-test was used to assess statistical significance. Data was also represented using appropriate diagrams like bar diagram.

Categorical outcome:

The association between VAP and no VAP and age group, gender, Elective / Emergency, Tracheal Aspirate, Culture, Grams Stain, CPIS Score, outcome, was assessed by cross tabulation and comparison of percentages. Chi square test was used to test statistical significance. Data was also represented using appropriate diagrams like bar diagram.

P value < 0.05 was considered statistically significant.

IBM SPSS version 21 was used for statistical analysis.

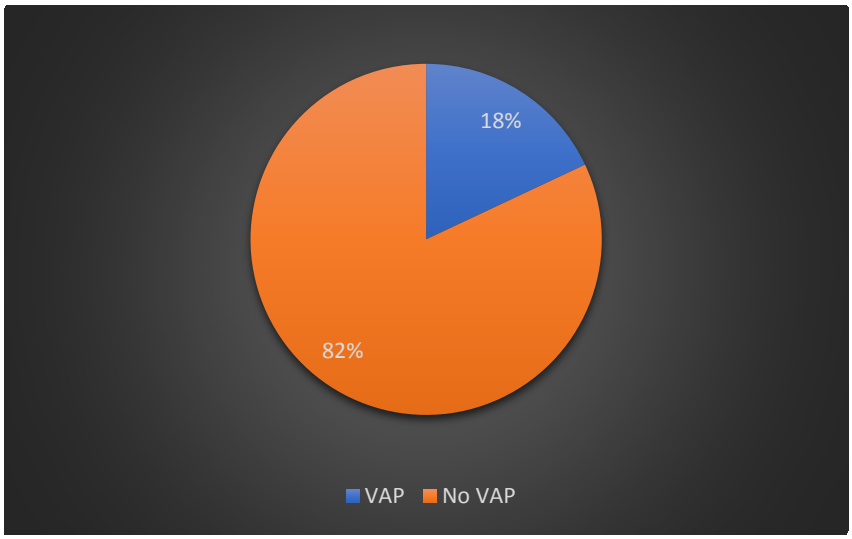
OBSERVATIONS AND DISCUSSION

1) INCIDENCE OF VAP : 18%

Table 1Descriptive analysis of VAP in study population

Early / Late VAP	Frequency	Percentage
VAP	9	18%
No VAP	41	82%
Total	50	100%

Figure 1: Pie chart of VAP in study population



Patients admitted in medical IMCU of GMKMCH were analysed in our study. 9 patients developed VAP and 41 patients did not develop VAP. The overall incidence of VAP in medical IMCU of GMKMCH according to this study is 18 %.

2) TYPES OF VAP

Table 2: Descriptive analysis of early and late VAP in study population

VAP	Frequency	Percentage
Early VAP	3	33.3%
Late VAP	6	66.7%
Total	9	100%

Figure 2: Pie chart of early / late VAP in study population

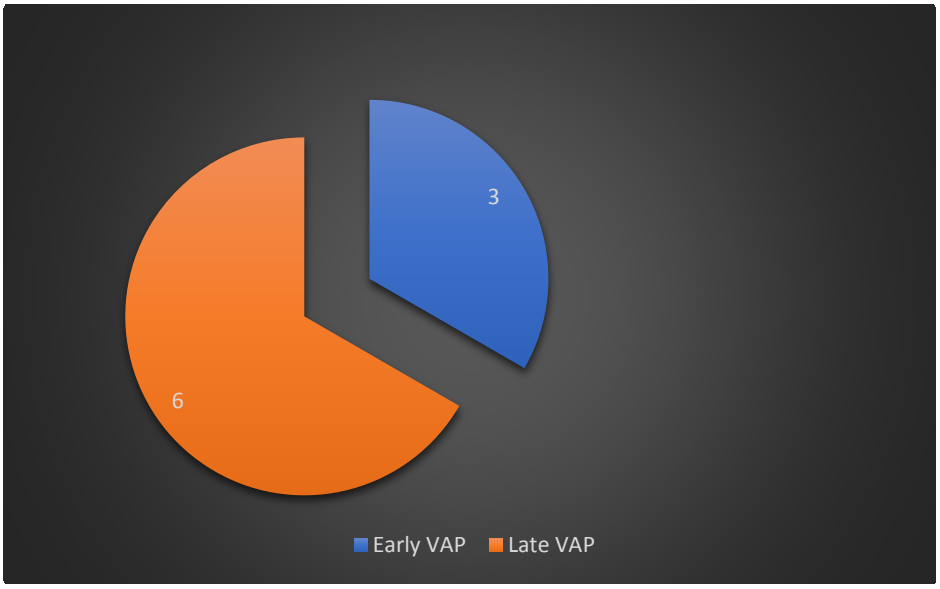
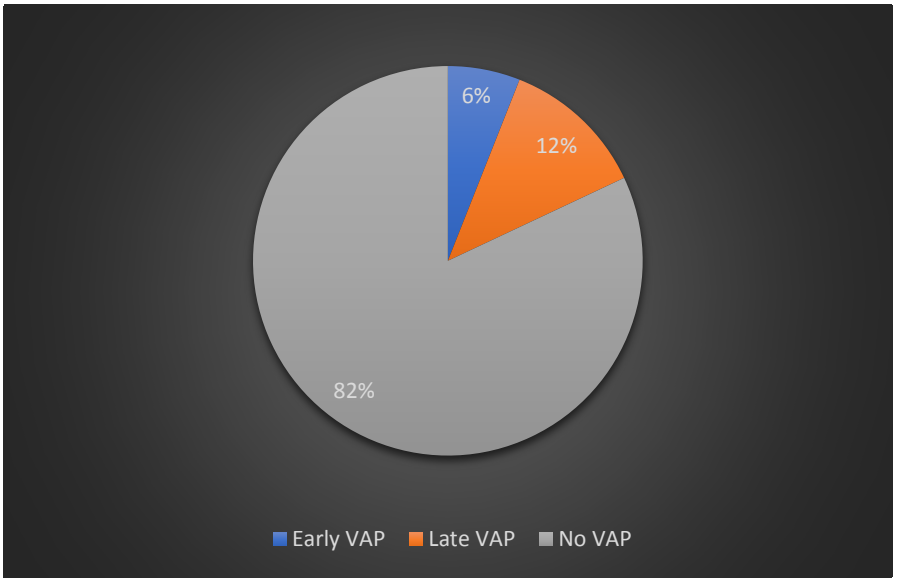


Table 9: Descriptive analysis of early / late VAP in study population (N=50)

Early / Late VAP	Frequency	Percentage
Early VAP	3	6%
Late VAP	6	12%
No VAP	41	82%
Total	50	100%

Figure 5: Pie chart of early / late VAP in study population (N=50)



Among the 50 patients studied 41(82%) had no VAP and 9(18%) had VAP. Among the 9 who developed VAP 3(6%) had early VAP and 6(12%) had late VAP ie early VAP was 33.3% and late VAP 66.7% respectively.

3) AGE DISTRIBUTION

Table 3: Comparison of age group with VAP

Age group	VAP		Total
	VAP	No VAP	
<30	4 (44.4%)	15 (36.6%)	19 (38%)
31- 60	4 (44.4%)	22 (53.7%)	26 (52%)
>60	1 (11.1%)	4 (9.8%)	5 (10%)
Total	9 (100%)	41 (100%)	50 (100%)
Chi Square = 0.254			
P value = 0.881 (Insignificant)			

Figure 3: Stacked bar chart of age group with VAP

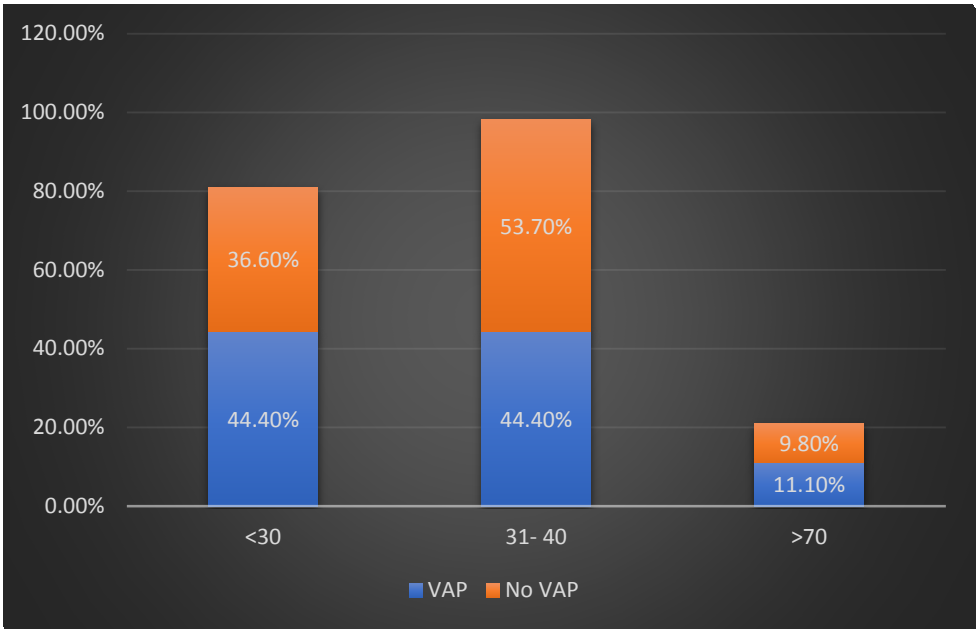
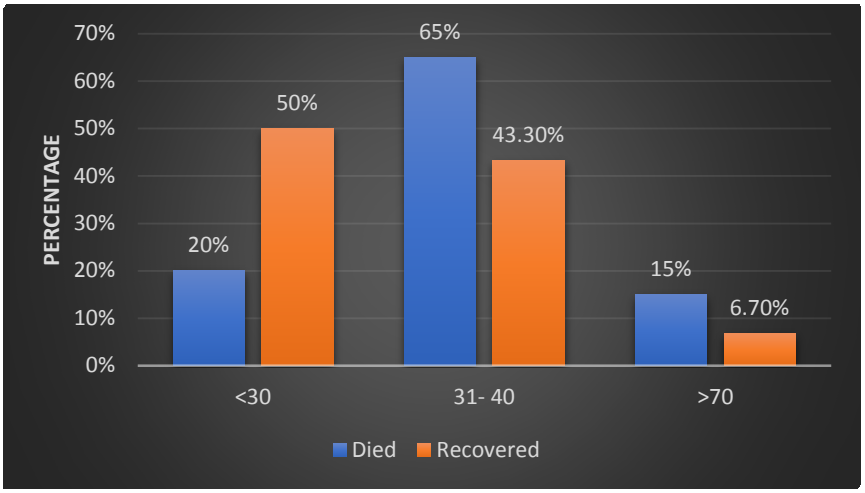


Table 4: Comparison of age group with outcome

Age group	Outcome		Total
	Died	Recovered	
<30	4 (20%)	15 (50%)	19 (38%)
31- 60	13 (65%)	13 (43.3%)	26 (52%)
>60	3 (15%)	2 (6.7%)	5 (10%)
Total	20 (100%)	30 (100%)	50 (100%)
Chi square test =4.76			
P value = 0.093 (Insignificant)			

Figure 4: Cluster bar chart of age group with outcome



The frequency of VAP in this study is 9 and the age wise distribution is as follows:

<30 years - 4(44.4%)

31-60 years - 4(44.4%)

>60 years - 1 (11.1%)

The higher incidence of VAP is seen in the younger age group less than 30 years and 31 to 60 years.

4) SEX DISTRIBUTION

Table 6: Comparison of gender with VAP

Gender	VAP		Total
	VAP	No VAP	
Male	8 (88.97%)	22 (53.7%)	30 (60%)
Female	1 (11.1%)	19 (46.3%)	20 (40%)
Total	9 (100%)	41 (100%)	50 (100%)
Chi square test =3.82			
P value = 0.051 (Significant)			

Figure 5: Stacked bar chart of gender with VAP

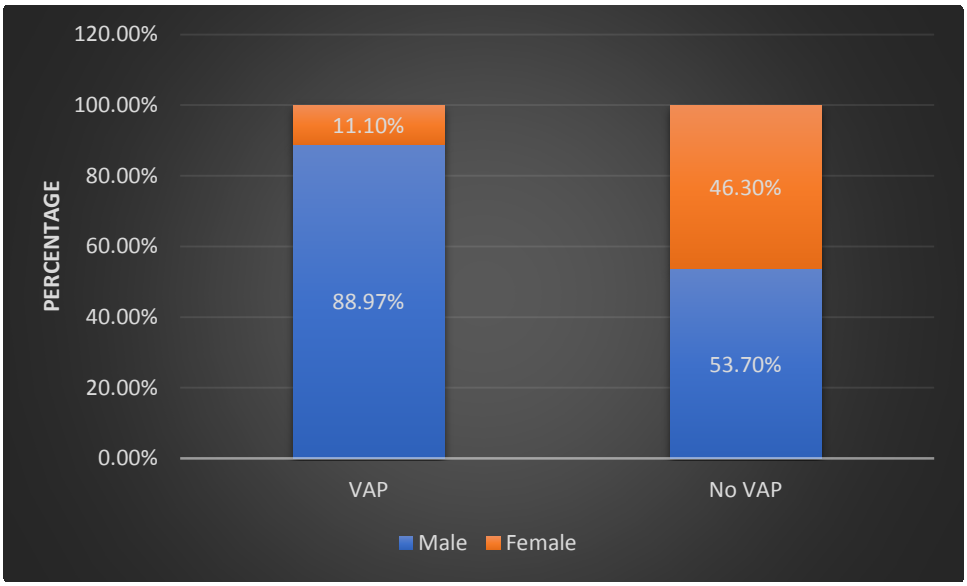
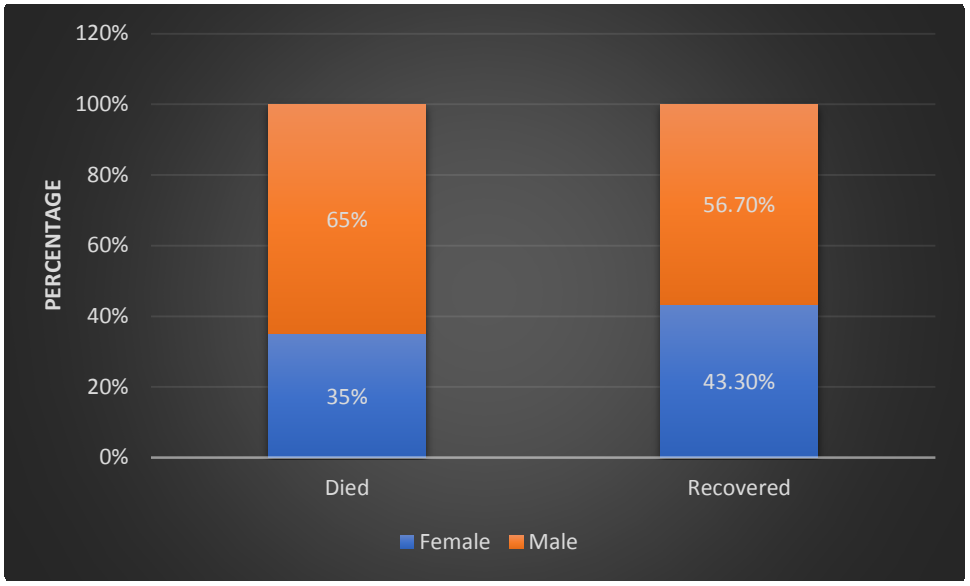


Table 5: Comparison of gender with outcome

Gender	Outcome		Total
	Died	Recovered	
Male	13 (65%)	17 (56.7%)	30 (60%)
Female	7 (35%)	13 (43.3%)	20 (40%)
Total	20 (100%)	30 (100%)	50 (100%)
Chi square test =0.347			
P value = 0.556 (Insignificant)			

Figure 5: Stacked bar chart of gender with outcome

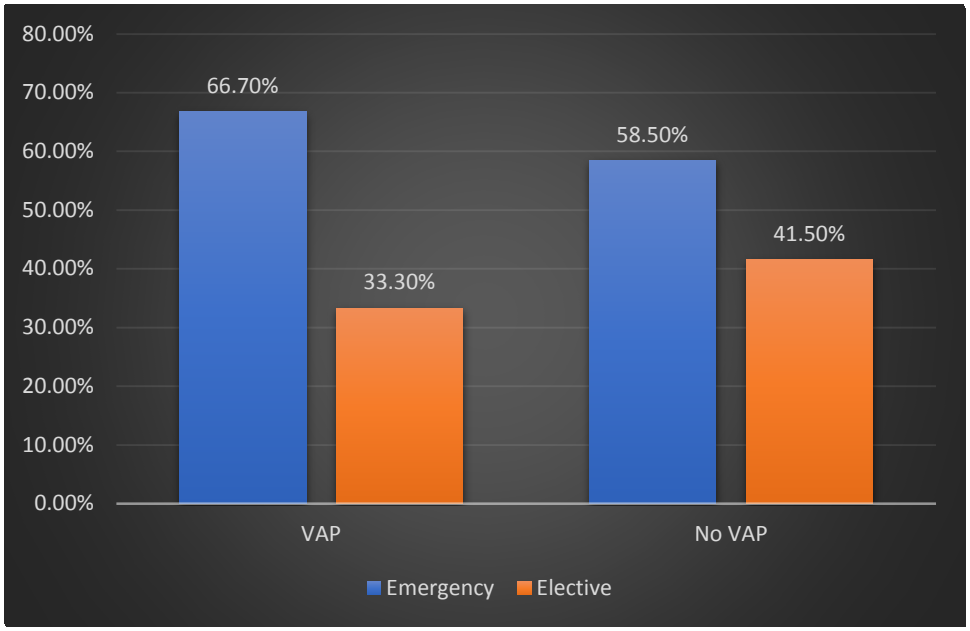


In our hospital among patients who developed VAP males were 8 (88.97%) and 1 was a female (11.1%). This may be due to increased number of male patients being admitted.

5) ELECTIVE VERSUS EMERGENCY INTUBATION

Table : Comparison of Emergency/ Elective with VAP

	VAP	No VAP	Total
Emergency	6 (66.7%)	24 (58.5%)	30 (60%)
Elective	3 (33.3%)	17 (41.5%)	20 (40%)
Total	9 (100%)	41 (100%)	50 (100%)
Chi Square – 0.203			
P value – 0.052			

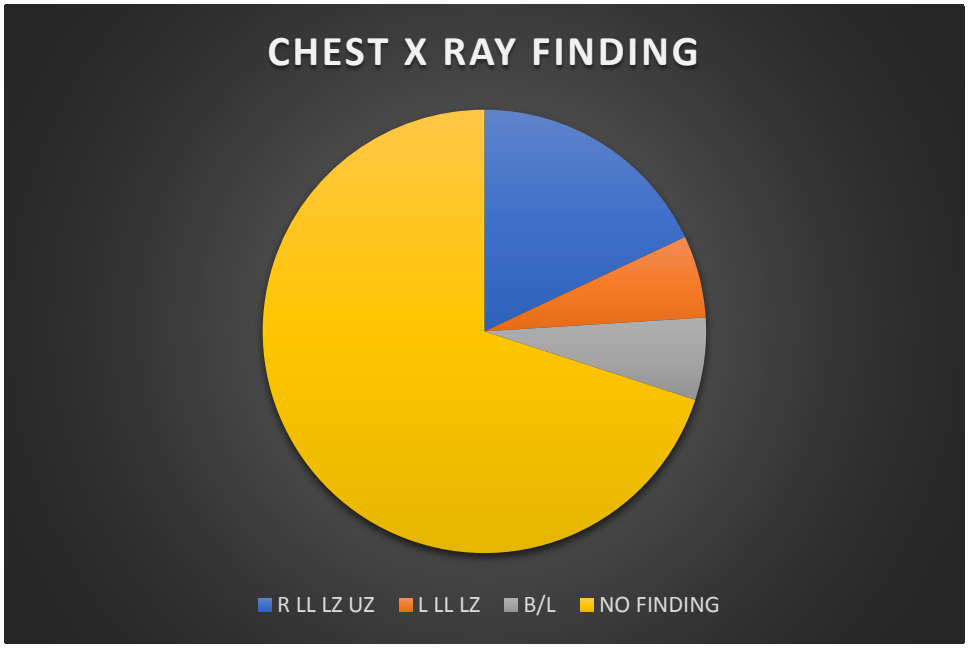


In this study 30 patients underwent emergency intubation and 20 patients underwent elective intubation. The frequency of VAP among emergency and elective intubation is 6(66.7%) and 3(33.3%) respectively. The method of intubation ie emergency intubation was found to have significant association with the development of VAP (p=0.052).

6) CHEST XRAY FINDINGS

Table 9: Descriptive analysis of CHEST X- RAY in study population (N=50)

CHEST X- RAY	Frequency	Percentage
(R) LL, LZ, UZ	9	18%
(L) LL, LZ	3	6%
B/L LL, Pl	3	6%
No Findings	35	70%
Total	50	100.0



Among the 50 patients studied 35 patients(70%) had a normal chest X ray. 3 patients(6%) had infiltration in the left lung , 9 patients(18%) had right lung infiltrates. 3(6%) of them had bilateral infiltrates.

7) MICROBIOLOGICAL PROFILE

Table 10: Descriptive analysis of tracheal aspirate in study population (N=50)

Tracheal aspirate	Frequency	Percentage
Purulent	10	20%
Non purulent	40	80%
Total	50	100%

Figure 7: Pie chart of tracheal aspirate in study population (N=50)

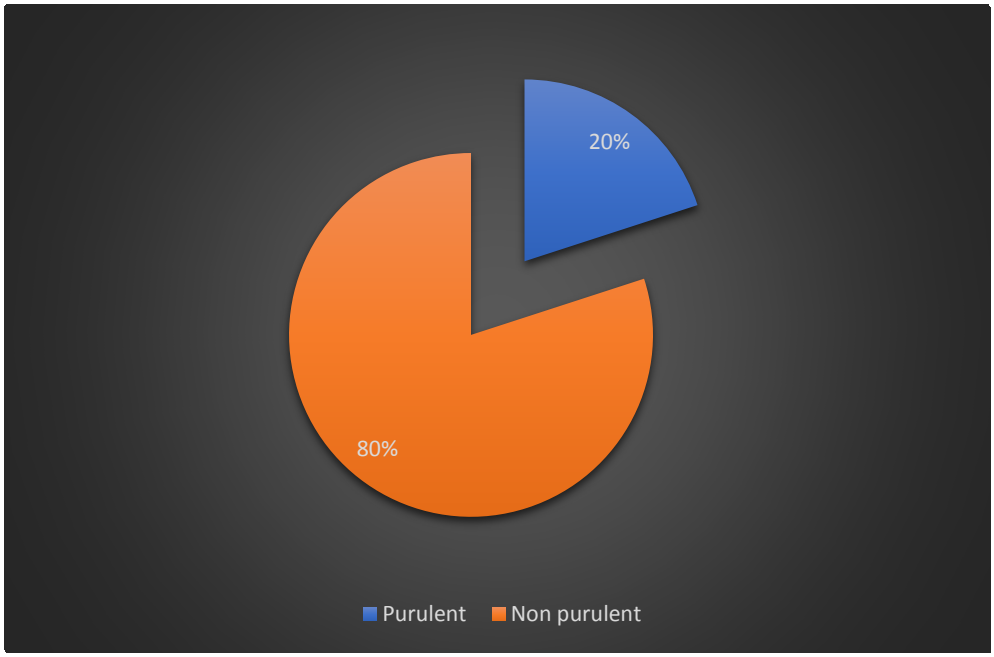


Table 11: Comparison of tracheal aspirate with VAP

Tracheal Aspirate	VAP		Total
	VAP	No VAP	
Non Purulent	0 (0%)	40 (97.6%)	40 (80%)
Purulent	9 (100%)	1 (2.4%)	10 (20%)
Total	9 (100%)	41 (100%)	50 (100%)
Chi square test = 43.90			
P value = <0.001 (Significant)			

Figure 5: Bar chart of tracheal aspirate with VAP

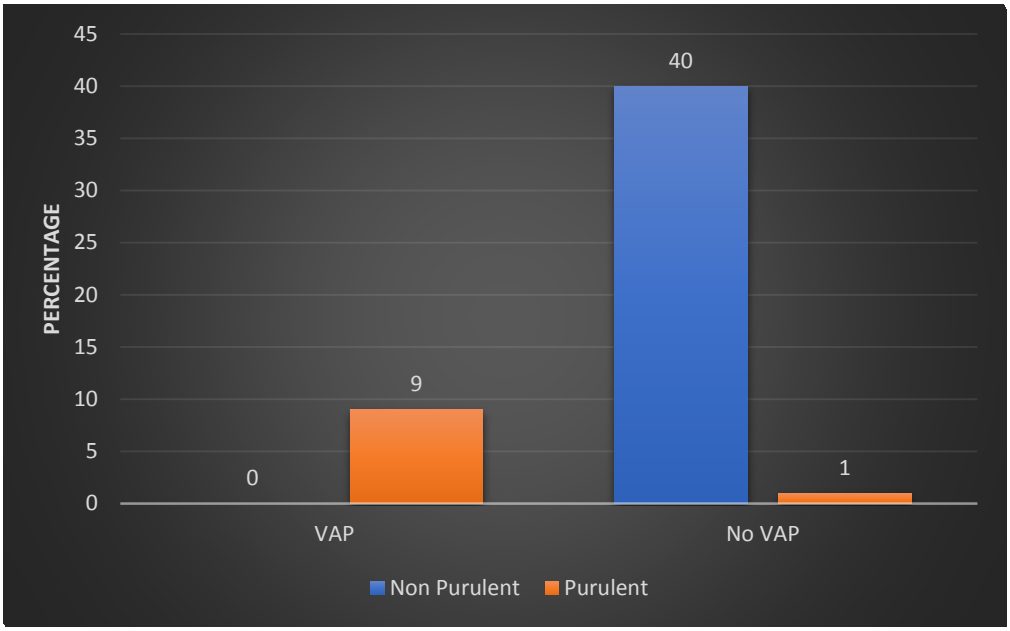


Table 12: Descriptive analysis of grams stain in study population (N=9)

Grams Stain	Frequency	Percentage
GNB	8	88.9%
GPC	1	11.1%
Total	9	100%

Figure 7: Pie chart of grams stain in study population (N=9)

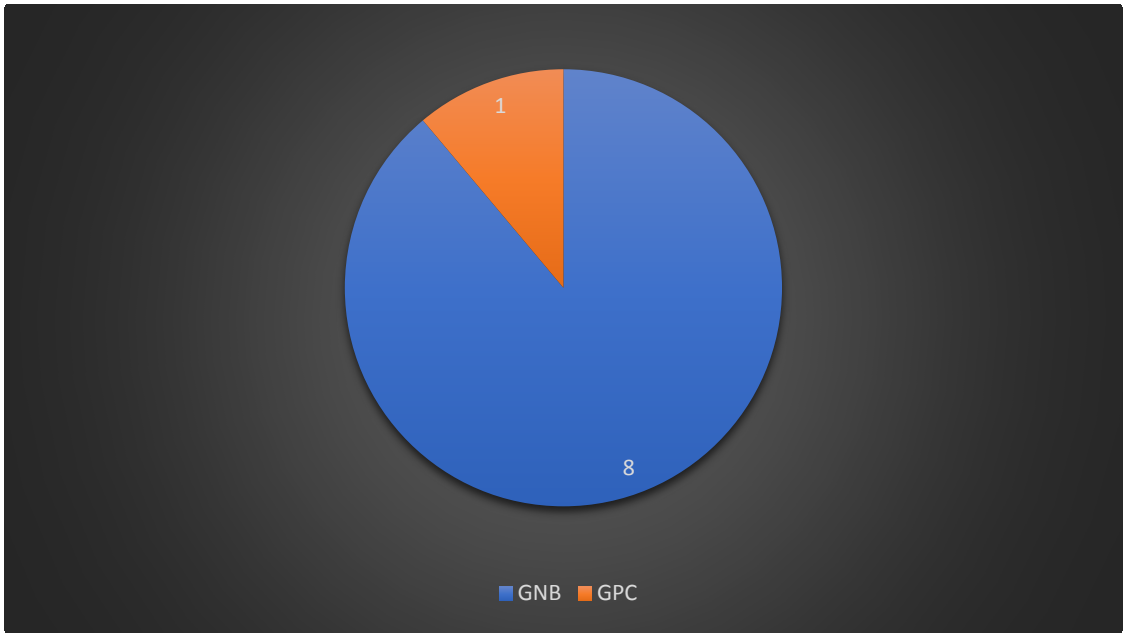
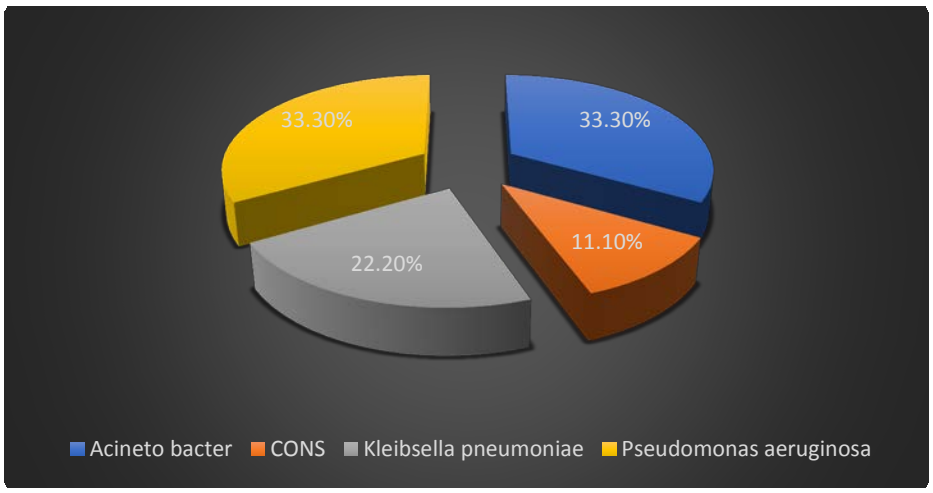


Table 13: Descriptive analysis of culture in study population

Culture	Frequency	Percentage
Acineto bacter	3	33.3%
CONS	1	11.1%
Kleibsella pneumoniae	2	22.2%
Pseudomonas aeruginosa	3	33.3%
Total	9	100%

Figure 8: Pie chart of culture in study population



Our study showed that out of the 9 cases of VAP, 8 (88.9%) were due to gram negative organisms and 1 (11.1%)was due to gram positive organism. The organisms identified in culture were as follows:

• Acinetobacter species	-	33.3% (3)
• Klebsiella pneumoniae	-	22.2% (2)
• Pseudomonas aeruginosa	-	33.3% (3)
• Coagulase negative Staphylococcus	aureus(CONS) -	11.1% (1).

8) CPIS SCORE AND ITS CORELATION

Table 15: Descriptive analysis of CPIS score in study population (N=50)

CPIS Score	Frequency	Percentage
≤6	43	86%
>6	7	14%
Total	50	100%

Figure 9: Pie chart of CPIS Score in study population (N=50)

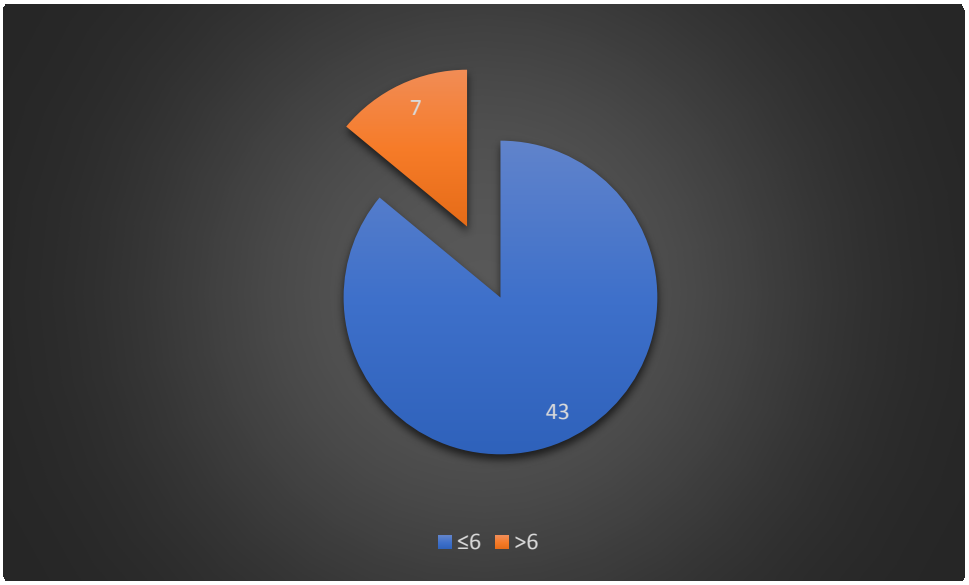
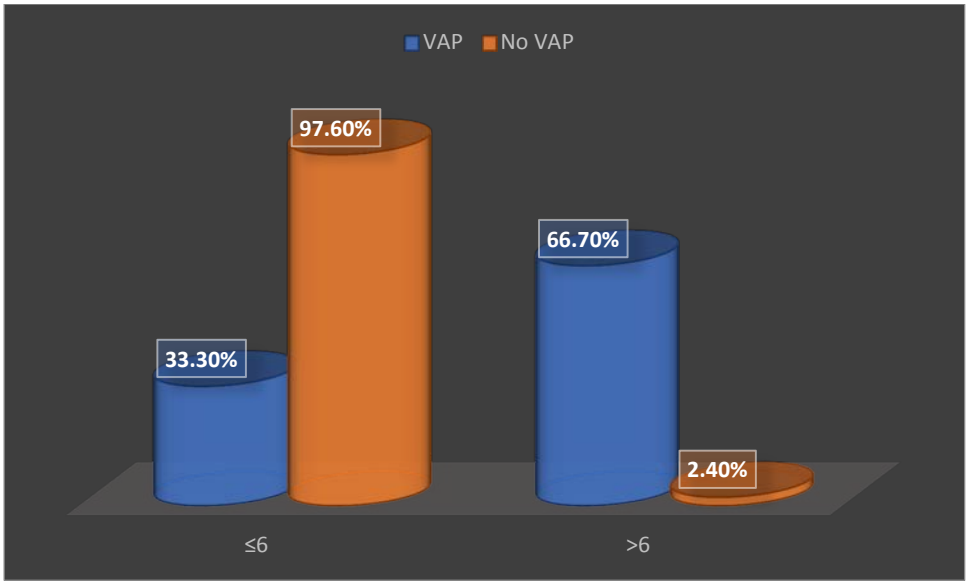


Table 16: Comparison of CPIS score with VAP

VAP	CPIS Score		Total
	≤6	>6	
VAP	3 (33.3%)	6 (66.7%)	9 (100%)
No VAP	40 (97.6%)	1 (2.4%)	41 (100%)
Total	43 (100%)	7 (100%)	50 (100%)
Chi square test =25.28			
P value = <0.001 (Significant)			

Figure 10: Bar chart of CPIS score with VAP



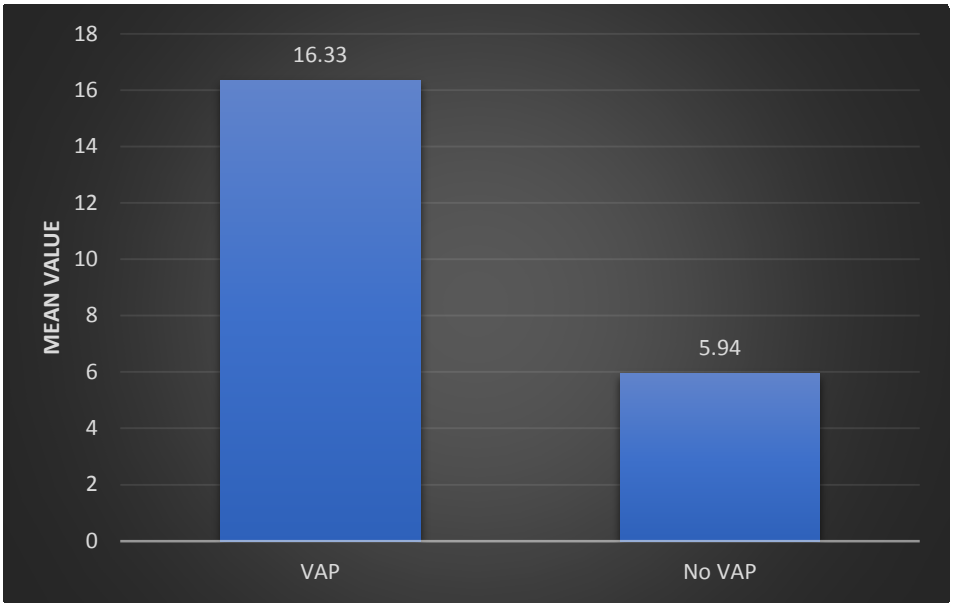
CPIS Scores vary between 0 and 12 . CPIS score of more than 6 is most commonly consistent with diagnosis of VAP. In our study among the 9 cases of VAP , 6 (66.7 %) had a score more than 6 and 3 (33.3%) had a score of 6. CPIS score o f more than 6 had a significant association with the diagnosis of VAP (p<0.001) according to chi square test in our study.

9) DURATION OF VENTILATION AND VAP ASSOCIATION

Table 17: Comparison of duration of ventilation (days) between VAP

	VAP		Unpaired t test
	VAP	No VAP	
Duration of ventilation (days)	16.33 ± 5.94	5.94 ± 5.38	<0.001

Figure 11: Bar chart of duration of ventilation (days) between VAP



Among the 9 cases of VAP ,6 patients were on mechanical ventilation for more than a week and 3 patients were on ventilator for less than a week. In our study there is a significant association between duration of mechanical ventilation and the development of VAP ($p<0.001$). The mean duration of mechanical ventilation was found to be 16 days in case of patients with VAP and 5 days in case of patients without VAP.

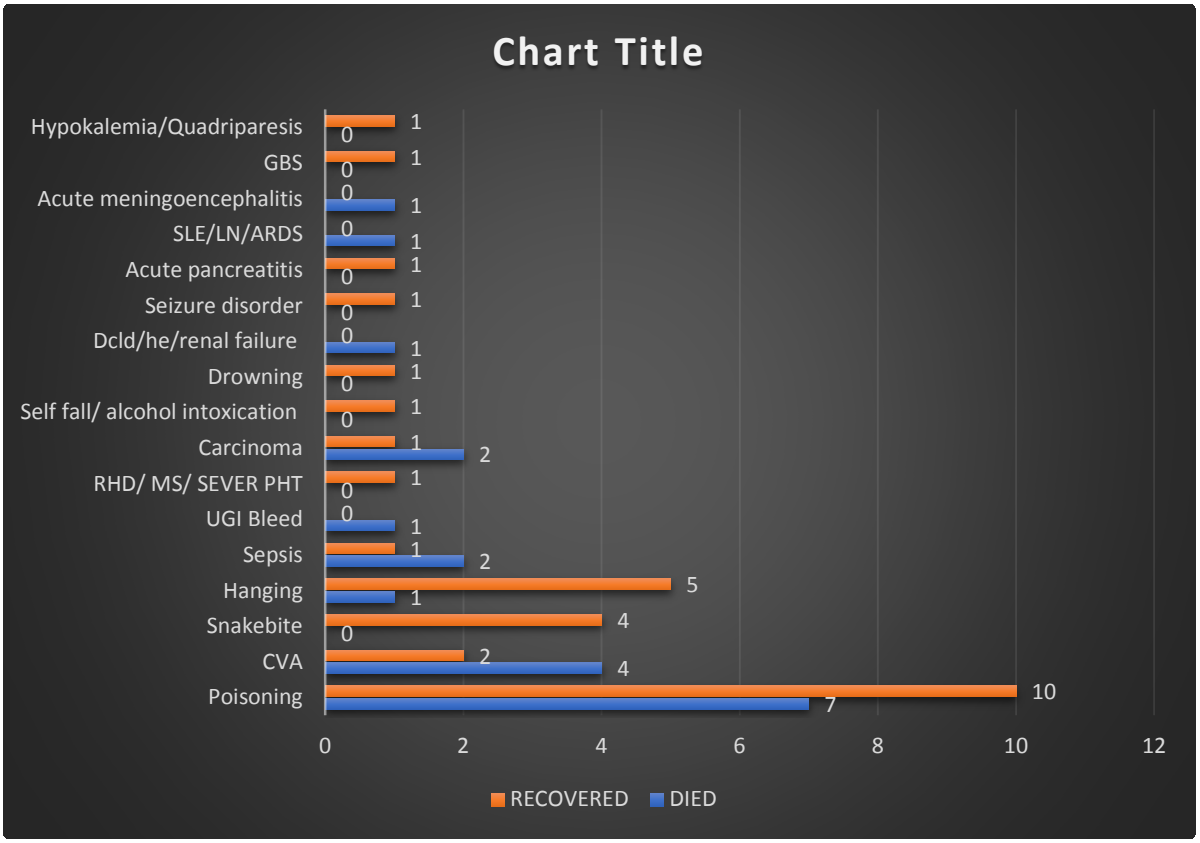
10) PRIMARY DIAGNOSIS AND VAP

Table : Comparison of primary diagnosis with VAP

DIAGNOSIS	CASES	VAP
POISONING	17	3
CVA	6	2
SNAKEBITE	4	1
HANGING	6	1
SEPSIS	3	0
UGI BLEED	1	1
RHD/ MS/ SEVER PHT	1	1
CARCINOMA	3	0
SELF FALL/ ALCOHOL INTOXICATION	1	0
DROWNING	1	0
DCLD/HE/RENAL FAILURE	1	0
SEIZURE DISORDER	1	0
ACUTE PANCREATITIS	1	0
SLE/LN/ARDS	1	0
ACUTE MENINGOENCEPHALITIS	1	0
GBS	1	0
HYPOKALEMIA/QUADRIPARESIS	1	0
Total	50	9
P value	0.636	

Table : Comparison of diagnosis with outcome

DIAGNOSIS	CASES	DIED	RECOVERED
Poisoning	17	7	10
CVA	6	4	2
Snakebite	4	0	4
Hanging	6	1	5
Sepsis	3	2	1
UGI Bleed	1	1	0
RHD/ MS/ SEVER PHT	1	0	1
Carcinoma	3	2	1
Self fall/ alcohol intoxication	1	0	1
Drowning	1		1
Dcld/he/renal failure	1	1	0
Seizure disorder	1	0	1
Acute pancreatitis	1	0	1
SLE/LN/ARDS	1	1	0
Acute meningoencephalitis	1	1	0
GBS	1	0	1
Hypokalemia/Quadriparesis	1	0	1
Total	50	20	30
P value	0.359		



In our study it is observed that there is no significant correlation between the primary diagnosis and development of VAP, P value is 0.636. Poisoning especially OPC poisoning is observed to be the most common diagnosis requiring IMCU admission and hence an apparent increase in the development of VAP among poisoning patients which is statistically insignificant. This may be due to the associated factors like need for prolonged ventilation, the increased secretions in OPC poisoning patients.

11) OUTCOME AND VAP

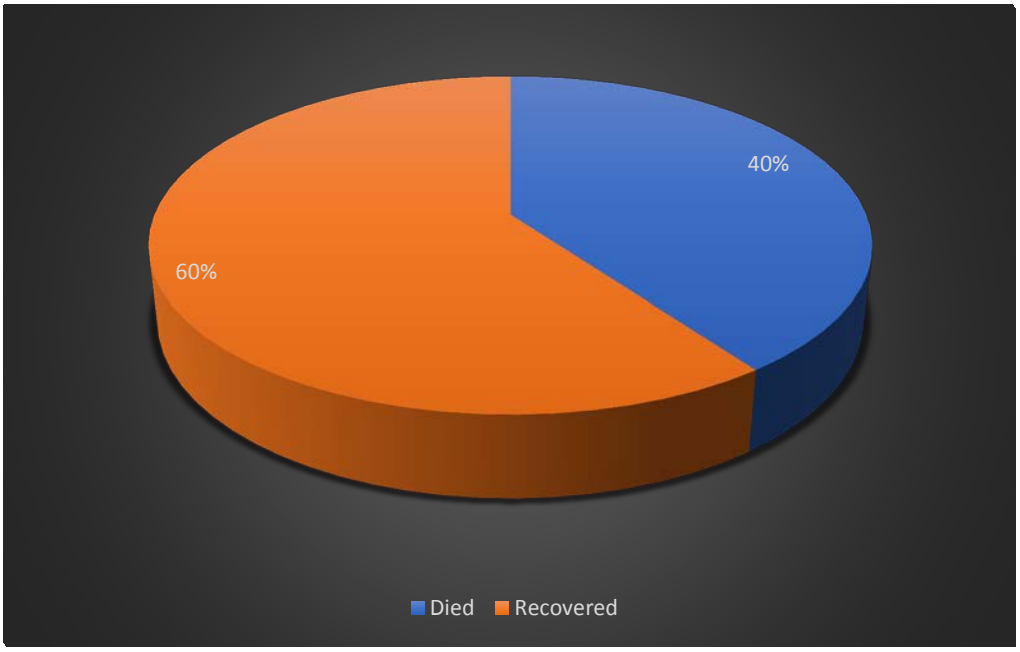
Table 18: Comparison of outcome with VAP

VAP	Outcome		Total
	Died	Recovered	
VAP	3 (33%)	6 (66.7%)	9 (100%)
No VAP	17 (41.5%)	24 (58.5%)	41 (100%)
Total	20 (100%)	30 (100%)	50 (100%)
Chi square test =2.287			
P value = 0.319 (Insignificant)			

Table 20: Descriptive analysis of outcome in study population (N=50)

	Frequency	Percentage
Died	20	40%
Recovered	30	60%
Total	50	100%

Figure 12: Pie chart of outcome in study population (N=50)



Among the 9 cases of VAP:

- 3 - died (33.3%)
- 6- recovered (66.67%)

The mortality rate in our study was observed to be 33% in case of patients with VAP and 41.5% in case of patients in NO VAP category . According to our study there is no statistical significance between the development of VAP and mortality as the outcome not only depends on the development of VAP but multifactorial and depends on various other factors like the primary diagnosis , underlying risk factors , age of the patient and many others.

DISCUSSION

(1) INCIDENCE OF VAP : 18%

50 patients admitted in medical IMCU of GMKMCH were analysed in our study. 9 patients developed VAP and 41 patients did not develop VAP. The overall incidence of VAP in medical IMCU of GMKMCH according to this study is 18 %. The prevalence of VAP worldwide may vary between 6 and 52 cases per 100 patients which depends on the population studied. In an ICU on any given day about 10% of patients may develop nosocomial pneumonia, especially VAP in the majority of cases. In a similar study conducted in West Bengal , India by Kalidas et al also had similar incidence of 20%. Another study done in northern India by Sanjay et al also had an incidence of 31.77%. A study Snigdha Rashmi et al conducted in Mysore , Karnataka had an incidence of 34.8%.

	KALIDAS ET AL	SANJAY ET AL	SNIGDHA ET AL	**OUR STUDY**
INCIDENCE	20%	31.7%	34.8%	**18%**

(2) TYPES OF VAP

Early onset VAP is defined as pneumonia which occurs after 2 days of mechanical ventilation but within 4 days of mechanical ventilation whereas late onset VAP is defined as pneumonia occurring 4 days after intubation. Among the 50 patients studied 41(82%) had no VAP and 9(18%) had VAP. Among the 9 who developed VAP 3(6%) had early VAP and 6(12%) had late VAP ie early

VAP was 33.3% and late VAP 66.7% respectively. The probability of acquiring VAP increases with the duration of mechanical ventilation. It has been found that risk for VAP increased from 5% in patients receiving one day of mechanical ventilation to 68.8% in patients receiving more than 30 days. Similar results were seen in other studies also like in Hina gadani et al published in the Indian journal of anaesthesia where incidence of early onset VAP was 27% and late VAP 73%. Other studies have also reported similar incidence of early onset VAP being more than the late onset VAP.

	HINA GADANI ET AL	**OUR STUDY**
EARLY VAP	27%	**33.3%**
LATE VAP	73%	**66.7%**

(3) AGE DISTRIBUTION

The frequency of VAP in this study is 9 and the age wise distribution is as follows:

<30 years - 4(44.4%)

31-60 years - 4(44.4%)

>60 years - 1 (11.1%)

The higher incidence of VAP is seen in the younger age group less than 30 years and 31 to 60 years. This can be attributed to more number of patients getting admitted in IMCU in this age group and undergoing mechanical ventilation . Due to the clustering of cases there is an apparent increase in the

incidence of VAP in the younger age group. Thus there is no significance in the age group and VAP incidence as the p value is 0.881 which is statistically insignificant.

(4) GENDER DISTRIBUTION

In our hospital among patients who developed VAP males were 8 (88.97%) and 1 was a female (11.1%). This may be due to increased number of male patients being admitted. The number of males included in study were 30 and the number of female included were 20 respectively. It is also observed that poisoning , the primary diagnosis most associated with development of VAP was more among males than the number of females admitted for poisoning.

(5) EMERGENCY VERSUS ELECTIVE INTUBATION :

In this study 30 patients underwent emergency intubation and 20 patients underwent elective intubation. The frequency of VAP among emergency and elective intubation is 6(66.7%) and 3(33.3%) respectively. The method of intubation ie emergency intubation was found to have significant association with the development of VAP (p=0.052). In case of emergency intubation there may be slack in the usual strict aseptic precautions due to the emergency situation. In case of elective intubation meticulous prior arrangements necessary for smooth intubation may have influenced in the statistical significance arrived between the emergency intubation being associated statistically significant with development of VAP .

(6) CHEST X RAY FINDINGS

The chest X-ray were normal in 35 of the 50 patients evaluated (70 %). Three patients (6%) had infiltrates in the left lung, while nine patients (18%) had infiltrates

in the right lung. Bilateral infiltrates were found in 3(6%) of them. Because aspiration is the most prevalent precipitating mechanism for VAP, previous research have revealed that it frequently involves the posterior right lower lobe. VAP commonly includes the posterior right lower lobe, according to autopsy research by Marquette C.H, M.C.Copin, F.Saulnier, D.Mathieu, A.Durocher, F.Wallet, R.Neviere, P.Ramon, and A.B.Tonnel in 1995.

(7) MICROBIOLOGICAL PROFILE

According to our findings, 8 (88.9%) of the 9 instances of VAP were caused by gram negative organisms, whereas 1 (11.1%) was caused by gram positive organisms. The following organisms were found in culture:

- Acinetobacter species – 3 (33.3%)
- Pseudomonas aeruginosa – 3 (33.3 %)
- Klebsiella pneumoniae – 2 (22.2 %)
- Coagulase negative staphylococcus aureus (CONS) – 1 (11.1 %)

Acinetobacter baumannii (37.63 %), Klebsiella pneumoniae (36.55 %), pseudomonas aeruginosa, and staphylococcus aureus were found in a similar study conducted in New Delhi by Shruvanu Ghosh, Amit Dhamija, Dhebasish Dhar, Arup Basu, and Neeraj Goel and published in the European respiratory journal. Distinct studies had different microbiological profiles, so developing a unique microbiological profile and antibiogram for each IMCU is critical.In contrast to community-acquired pneumonia, where Streptococcus pneumonia is the most common cause, this study demonstrates a higher occurrence of MDR organisms as a cause of VAP. In another

study done in Maharashtra by Babasaheb et al 36% had pseudomonas followed by 26% with Acinetobacter infection and 14% with staphylococci infection. Yet another study by Ashu sara mathai et al showed 53.2% of Acinetobacter infection followed by klebsiella 15.6% and pseudomonas 12.8 %. Thus the microbiological profile of each institute , each region and even each ICU is different. Hence an institute specific , to be precise ICU specific microbiological profile and antibiogram to be established.

ORGANISM	GHOSH ET AL	BABASAHEB ET AL	ASHU ET AL	OUR STUDY
ACINETOBACTER	37.63%	26%	53.2%	**33.3%**
PSEUDOMONAS		36%	12.8%	**33.3%**
KLEBSIELLA	36.55%		15.6%	**22.2%**
STAPHYLOCOCCI		14%		**11.1%**

(8) CPIS SCORE AND ITS CORRELATION

The Clinical Pulmonary Infection Score (CPIS) is a numerical value that combines clinical, microbiological, and radiographic information to predict the presence or absence of VAP. The CPIS score ranges from 0 to 12. A CPIS score of more than 6 is most often associated with a VAP diagnosis. 6 (66.7 %) of the nine cases with VAP in our study had a score of higher than 6, whereas 3 (33.3 %) had a score of less than 6. According to the chi square test, a CPIS score of higher than 6 indicated a significant connection with the diagnosis of VAP ($p < 0.001$). To make the diagnosis of VAP easier, Pugin et al devised CPIS scoring. In terms of diagnosing VAP, it has a sensitivity of 93 percent and a specificity of 100 percent. According to a subsequent study, the CPIS scoring system has a sensitivity of 72 to 77 percent and a

specificity of 42 to 85 % in diagnosing VAP. According to a study published in International journal of infection control by Nasia safdar et al done in the University of Wisconsin hospital , USA the original and modified CPIS score were compared with CDC NHSN Centre for Disease Control National Healthcare Safety Network surveillance definition of VAP. Both the original and modified CPIS was found to have a sensitivity of 95% and specificity of 95% as well.

(9) DURATION OF MECHANICAL VENTILATION AND ASSOCIATION WITH VAP

The risk of VAP is greatest during the first five days of mechanical ventilation, with an average time of 3.3 days between intubation and VAP development. The duration of mechanical ventilation is observed to be directly related to the occurrence of VAP. After 5 to 10 days of mechanical ventilation, the risk of VAP lowers to 2% per day, then to 1% per day after that. Similarly, Apostolopoulou et al found an elevated risk of VAP during the first two weeks of mechanical ventilation. In our study 6 of the 9 patients with VAP had been on mechanical ventilation for more than a week, while 3 had been on it for less than a week. There is a statistical significance between the duration of mechanical ventilation and the development of VAP in our study ($p<0.001$).

It was discovered that the average duration of mechanical ventilation was 16 days in patients with VAP and 5 days in patients without VAP. In a similar study by Hina Gadani et al also the mean duration of ventilation for VAP patients was 19 days whereas for NON VAP patients it was 11 days. According to an Italian study

conducted with 724 ICU patients, the incidence of VAP increased from a mere 5% on day 1 to 69 % on day 30. Thus the duration of mechanical ventilation significantly increases the risk of developing VAP and as well the ICU stay duration.

(10) PRIMARY DIAGNOSIS AND VAP

In our study it is observed that there is no significant correlation between the primary diagnosis and development of VAP, P value is 0.636. Poisoning especially OPC poisoning is observed to be the most common diagnosis requiring IMCU admission and hence an apparent increase in the development of VAP among poisoning patients which is statistically insignificant. This may be due to the associated factors like need for prolonged ventilation, the increased secretions in OPC poisoning patients. The underlying risk factor especially diabetes mellitus has a significant association with the development of VAP but the primary diagnosis for which the patient is in admission doesnot raise the likelihood of VAP.

(11) FINAL OUTCOME AND VAP

Among the 9 cases of VAP:

- 3 - died (33.3%)
- 6- recovered (66.67%)

The mortality rate in our study was observed to be 33% in case of patients with VAP and 41.5% in case of patients in NO VAP category . According to our study there is no statistical significance between the development of VAP and mortality as the outcome not only depends on the development of VAP but multifactorial and depends on various other factors like the primary diagnosis , underlying risk factors , age of the patient and many others. Tejerina et al who studied more than two thousand

patients with VAP and found that although VAP was associated with a significant increase in hospital stay it did not influence the ICU mortality. In another study by Hina Gadani et al the mortality rate in VAP group was 54.05% as compared to 41.2% in NON VAP group but this was not statistically significant. In a similar study by Ashu Sara Mithai et al the mortality rate was 68.4% and 61.3% in VAP group and NON VAP group respectively.

	HINA ET AL	ASHU ET AL	**OUR STUDY**
VAP	54.05%	68.4%	**33%**
NO VAP	41.2%	61.3%	**41.5%**

CONCLUSION

We arrive at the following conclusions

- The Incidence of VAP in our study is directly proportional to the duration of mechanical ventilation . To reduce the morbidity and mortality associated with mechanical ventilation, the length of ventilation must be reduced, which can be accomplished by devising an appropriate weaning procedure and titrating the sedative regimes according to the patient's demands.

- The other factors like age , gender, the primary diagnosis donot have a statistically significant association with the development of VAP.

- Patients should be placed in semi-recumbent position with the head end elevated to 45° as the supine positioning promotes aspiration which is a strong underlying factor for the development of VAP.

- Aspiration is a major proven precipitating factor for development of VAP and Diabetes mellitus is an important risk factor influencing the mortality and morbidity.

- Acinetobacter and Pseudomonas are the most common organism in our institution. Thus there is a increased incidence of MDR organisms involved in VAP unlike in CAP.

- Late-onset VAP which is more in incidence is observed to be associated with poor prognosis as compared to the early-onset VAP variety.

- The outcome ie the IMCU mortality is multifactorial and thus not all mortalities in IMCU could be attributed only to VAP.
- The major goals in VAP management are prompt and early diagnosis , appropriate antibiotics according to the organism and sensitivity followed by meticulous de-escalation according to the microbiological profile and clinical response of the patient.
- Prevention of VAP is of paramount importance and all measures aimed at prevention like educating the care givers regarding following the ventilator bundle are to be followed appropriately.

LIMITATION OF STUDY

LIMITATION OF OUR STUDY

- The major limitation and drawback of the study is the sample size. Better results could be obtained if a larger group was studied and followed up for a long period.
- The study is also conducted only in the medical ICU. The causative organisms and the antibiogram may be different in different ICU setups. Thus our result and observations cannot be generalized to the institute and region.
- The mortality or outcome of this study can be attributed to causes other than VAP. Hence accurate mortality outcome rates or attributable mortality due to VAP could not be measured.

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PROFORMA

NAME

AGE/SEX

IP No.

COMPLAINTS:

RISK FACTORS:

CAUSE FOR INTUBATION

DURATION OF MECHANICAL VENTILATION

TRAUMA AS A REASON FOR INTUBATION

ANTIBIOTIC USAGE IN THE PAST ONE WEEK

PERSONAL HISTORY:

SMOKER / ALCOHOLIC

VITAL SIGNS:

TEMPERATURE

PULSE RATE

BLOOD PRESSURE

ON EXAMINATION:

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

ABDOMEN

CENTRAL NERVOUS SYSTEM

INVESTIGATIONS:

TC

DC

HB

PLATELETS

CHEST X RAY ON ADMISSION

CHEST X RAY AFTER 48 HOURS

ORGANISMS GROWN IN ENDOTRACHEAL ASPIRATE

CLINICAL PULMONARY INFECTION SCORE

CRITERIA	CPIS SCORE	
	Day 0	Day 3
TEMPERATURE		
LEUKOCYTE COUNT		
PAO2/FIO2 RATIO		
CHEST X RAY		
CULTURE OF TRACHEAL ASPIRATE		

PATIENT CONSENT FORM

STUDY TITLE:

AN OBSERVATIONAL STUDY ON INCIDENCE , ETIOLOGY , RISK FACTORS , OUTCOME AND PREVENTION OF VENTILATOR ASSOCIATED PNEUMONIA IN IMCU PATIENTS ADMITTED IN GMKMCH , SALEM

DEPARTMENT OF GENERAL MEDICINE, GMKMCH SALEM

PARTICIPANT NAME: AGE: SEX:

O.P. NO:

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during and after medical procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethical committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study.

Time: Patient name;
Date: Signature / Thumb Impression of Patient:
Place Name and signature of the Investigator

<u>ஆராய்ச்சி ஒப்புதல் படிவம்</u>

ஆராய்ச்சி தலைப்பு:

பெயர்	:	தேதி	:
வயது	:	உள்நோயாளி எண்	:
பாலினம்	:	ஆய்வு சேர்க்கைஎண்	:

இந்த ஆய்வின் நோக்கம் மற்றும் விவரங்கள் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. இவ்வாய்வில் இருந்து நான் எந்த நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எனக்கு எந்த பாதிப்பும் இல்லை என்பதையும் தெளிவாக புரிந்து கொண்டேன்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆய்வின் போதோ என்னுடைய பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டார்கள் என்பதையும் அறிந்து கொண்டேன்.

இந்த ஆய்வில் எவ்வித நிர்பந்தமும் இன்றி எனது சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகின்றேன்.

நான் சுயநினைவுடனும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் சேர்த்துக்கொள்ள சம்மதிக்கின்றேன்.

ஆராய்சியாளர் ஒப்பம்

பங்கேற்பாளர் ஒப்பம்
(அ)
இடது பெருவிரல் ரேகை

MASTER CHARTS

S. No.	NAME	AGE	SEX	DIAGNOSIS	INDICATION	ELECTIVE / EMERGENCY	RISK FACTOR	CHEST X- RAY	TEMP	TLC	TRACHEAL ASPIRATE	GRAMS STAIN	CULTURE	CPIS SCORE	EARLY / LATE VAP	ABG	DURATION OF VENTILATION	OUTCOME
1	Sathish	30	M	OPC Poisoning	Resp failure	Emergency	No	(R) UZ Haziness	102 F	17500	Purulent	GNB	Acineto bacter	8	Late VAP	Met. Alkalosis	24 days	Recovered
2	Kuppusamy	77	M	DM / SHT / brainstem stroke	Poor gcs	Elective	DM/SHT	(R) Diffuse infiltrate	100 F	19600	Purulent	GNB	Kleibsella pneumoniae	8	Early VAP	Resp. acidosis	16 days	Recovered
3	Marakkal	64	F	DM/ACUTE CVA Massive infarct	Poor gcs	elective	DM	N	N	10500	Non Purulent		No growth	0	No VAP	N	3 days	Died
4	Shankar	35	M	Monocrotophos poisoning	Resp failure	Emergency		N	101 F	21000	Non Purulent		No growth	0	No VAP	Met. Alkalosis	6 days	Died
5	Rajesh	32	M	Alcohol intoxication / self fall	Resp failure	Emergency		N	N	11000	Non Purulent		No growth	0	No VAP	N	3 days	Recovered
6	kannan	22	M	Rat killer paste poisoning	Poor gcs	Elective		(R) LZ Consolidation	N	12300	Non Purulent		No growth	5	No VAP	Met. Alkalosis	9 days	Died
7	Pavithra	23	F	OPC Poisoning	Resp failure	Emergency		(R) LL Consolidation	100F	11100	Purulent		No growth	4	No VAP	N	10 days	Recovered
8	Dilipa	24	F	Neurotoxic snake bite	Resp failure	Emergency		N	N	15400	Non Purulent		no growth	0	No vap	N	3 days	recovered
9	Meena	20	F	Neurotoxic snake bite	Resp failure	Emergency		N	N	9400	Non Purulent		No growth	0	No VAP	N	3 days	recovered
10	Kumaravel	48	M	OPC Poisoning	Resp failure	Elective		N	99 F	15700	Non Purulent		No growth	2	No VAP	N	3 days	Died
11	Soundammal	65	F	DM/CAD /uro sepsis	Poor gcs	Elective	DM	B/L Pl. effusion	99 F	12000	Non Purulent		No growth	5	No VAP	N	3 days	Recovered
12	Nandhini	27	F	Neurotoxic snake bite	Resp failure	Emergency		(L) Diffuse infiltrate	100 F	16800	Purulent	GNB	Kleibsella pneumoniae	6	Early VAP	N	11 days	Recovered
13	Reginamary	44	F	DM/alprax poisoning	Poor gcs	Elective	DM	N	100 F	16900	Non Purulent		No growth	0	No VAP	N	4 days	Recovered
14	Kanagaraj	48	M	Acute CVA/ ICH	Poor gcs	Emergency	SHT/DM	(R) UZ infiltrate	98.6 F	16900	Purulent	GNB	Acinetobacter	6	Late VAP	N	20 days	Died
15	Revathi	30	F	OPC Poisoning	Resp failure	Emergency		N	102 F	17300	Non Purulent		No growth	2	No VAP	N	5 days	Recovered
16	Sugumar	54	M	Hanging/Alcohol intoxication/DM	Resp failure	Emergency	DM	(R) LL / Consolidation	101 F	6700	Non Purulent		No growth	3	No VAP	Met. Acidosis	5 days	Recovered
17	Afsar ali	78	M	DM / Sepsis/MODS	Poor gcs	Elective	DM	N	N	16900	Non Purulent		No growth	0	No VAP	Met. Acidosis	3 days	Died
18	Surya	21	M	Rat killer paste poisoning	Poor gcs	Elective		N	100 F	4700	Non Purulent		No growth	0	No VAP	Met. Alkalosis	18 days	Died
19	Vasanthi	52	F	SHT/Acute CVA/ICH	Poor gcs	Elective	SHT	N	101 F	8900	Non Purulent		No growth	2	No VAP	Met. Alkalosis	2 days	Died
20	Karunamoorthy	31	M	Pyrethroid poisoning	Resp failure	Emergency		N	104 F	14600	Non Purulent		No growth	4	No VAP	Met. Acidosis	3 days	Recovered
21	Ravichandran	40	M	Chlorpyrifos poisoning	Resp failure	Emergency		N	99 F	15400	Non Purulent		No growth	3	No VAP	N	4 days	Recovered
22	Praveen	49	M	OPC Poisoning (Monocrotophos)	Resp failure	Emergency	DM	(R) LL consolidation	101 F	15580	Purulent	GNB	Acinetobacter	7	Late VAP	N	17 days	Died
23	Kayal	52	F	DM/CKD/ICH	Poor gcs	Elective		N	100 F	10600	Non Purulent		No growth	0	No VAP	N	4 days	Died
24	Shankar	25	M	Near drowning	Resp failure	Emergency		B/L LL Haziness	101 F	10000	Non Purulent		No growth	5	No VAP	N	4 days	Recovered
25	Bharathi	22	F	Hanging	Resp failure	Emergency		(L) LL Consolidation	100 F	15400	Non Purulent		No growth	5	No VAP		5 days	Recovered

S. No.	NAME	AGE	SEX	DIAGNOSIS	INDICATION	ELECTIVE / EMERGENCY	RISK FACTOR	CHEST X- RAY	TEMP	TLC	TRACHEAL ASPIRATE	GRAMS STAIN	CULTURE	CPIS SCORE	EARLY / LATE VAP	ABG	DURATION OF VENTILATION	OUTCOME
26	Sabari	52	M	Hanging	Resp failure	Emergency		N	N	11000	Non Purulent		No growth	1	No VAP		2 days	Recovered
27	Mariyammal	25	F	Hanging	Resp failure	Emergency		N	N	10600	Non Purulent		No growth		No VAP	Resp. alkalosis	3 days	Recovered
28	Shiva	48	M	RHD/MS/Severe PHT/respiratory failure	Resp failure	Emergency	RHD/MS/ severe PHT	(L) LL consolidation	100˚F	17700	Purulent	GNB	Pseudomonas aeruginosa	7	Late VAP	Resp. alkalosis	22 days	Recovered
29	Divya barathi	23	F	Neurotoxic snake bite	Resp failure	Emergency		N	100˚F	21000	Non Purulent		No growth	3	No VAP	N	3	Recovered
30	Yuvaraj	20	M	Hanging	Resp failure	Emergency		(R) LL Consolidation	100˚F	16900	Purulent	GNB	Pseudomonas aeruginosa	7	Late VAP		11	Recovered
31	Elumalai	40	M	OPC poisoning/DM	Resp failure	Emergency	DM	N	99˚F	10400	Non Purulent		No growth	2	No VAP	N	3	Recovered
32	Malathi	66	F	DM/R Emphysematous pyelonephritis	Poor gcs	Elective	DM	N	100˚F	11000	Non Purulent		No growth	2	No VAP	Resp. alkalosis	4	Died
33	Valliappan	48	M	Ca lung with brain metastasis	Poor gcs	Elective	Malig nancy	N	102˚F	8900	Non Purulent		No growth	2	No VAP	N	7	Died
34	Kumar	48	M	OPC poisoning	Resp failure	Emaergency		N	102˚F	11600	Non Purulent		No growth	3	No VAP	N	4	Recovered
35	Savithri	57	F	DM / SHT/ CLL	Poor gcs	Emergency	DM /SHT Malignancy	N	102˚F	55500	Non Purulent		No growth	4	No VAP	Met. acidosis	19	Recovered
36	Murugan	38	M	OPC Poisoning	Resp failure	Emergency		N	102˚F	2700	Non Purulent		No growth	2	No VAP	Met. acidosis	15	Died
37	Rajalingam	21	M	OPC Poisoning	Resp failure	Emergency		N	N	3500	Non Purulent		No growth	0	No VAP	Resp. alkalosis	5	Recovered
38	Sivaraman	58	M	DCLD/HE/Renal failure	Poor gcs	Elective	DCLD	N	100˚F	11000	Non Purulent		No growth	2	No VAP	Met alkalosis	5	Died
39	Ramya	20	F	Seizure disorder/ status epilepticus	Poor gcs	Elective	Seizure disorder	N	99˚F	8500	Non Purulent		No growth	1	No VAP	N	4	Recovered
40	Jancy	21	F	SLE / LN / ARDS	Resp failure	Emergency	SLE	B/L LL Haziness	101˚F	19000	Non Purulent		No growth	6	No VAP	ARDS	5	Died
41	Subash	25	M	Carbamate poisoning	Resp failure	Emergency		(L) LZ Haziness	102˚F	14300	Purulent	GNB	Pseudomonas aeruginosa	8	Early VAP	N	6	Recovered
42	Ravi	28	M	DM / DKA / Acute pancreatitis	Poor gcs	Elective	DM	N	100˚F	9700	Non purulent		No growth	0	No VAP	Met. acidosis	4	Recovered
43	Abdul Jaleel	31	M	Acute meningo encephalitis	Resp failure	Emergency		N	100˚F	14200	Non Purulent		No growth	3	No VAP		3	Died
44	Mohanavel	45	M	OPC Poisoning (monocrotophos)	Resp failure	Emergency		N	98.6˚F	7300	Non Purulent		No growth	0	No VAP		3	Died
45	Srinivasan	50	M	(L) ICH / DM	Poor gcs	Elective	DM	N	99˚F	4800	Non Purulent		No growth	1	No VAP		3	Recovered
46	Elumalai	56	M	Chronic alcoholic/ UGI Bleed	Poor gcs	Elective		(R) LL Opacity	99˚F	11300	Purulent	GPC	CONS	6	Late VAP	Resp. alkalosis	20	Died
47	Raghu	43	M	GBS / autonomic dysfunction	Resp failure	Elective		N	99˚F	3800	Non Purulent		No growth	1	No VAP		5	Recovered
48	Anurekha	28	F	Hanging	Resp failure	Emergency		N	101˚F	15900	Non Purulent		No growth	8	No VAP	Met. Acidosis	29	Died
49	Selvi	31	F	Hypokalemia / Quadriparaesis	Resp failure	Elective		N	99˚F	25500	Non Purulent		No growth	3	No VAP		9	Recovered
50	Sangeetha	43	F	AML / SAH	s	Emergency	Malig nancy	N	102˚F	6300	Non Purulent		No Growth	2	No VAP	Met. Acidosis	8	Died