

**“COMPARISON OF VALIDITY OF SEVERITY SCORING  
SYSTEMS IN COMMUNITY ACQUIRED PNEUMONIA.”**

By

**Dr. UPHAR GUPTA**



DISSERTATION SUBMITTED TO THE SRI DEVARAJ URS ACADEMY  
OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

*In partial fulfilment of the requirements For the degree of*

**DOCTOR OF MEDICINE**

**IN**

**General Medicine**

**Under the guidance of**

**Dr. VIDYASAGAR C.R., MD**

**Associate Professor**



**DEPARTMENT OF GENERAL MEDICINE,  
SRI DEVARAJ URS MEDICAL COLLEGE  
TAMAKA, KOLAR-563101**

**MAY 2016**

## **DECLARATION BY THE CANDIDATE**

I hereby declare that this dissertation entitled “**COMPARISON OF VALIDITY OF SEVERITY SCORING SYSTEMS IN COMMUNITY ACQUIRED PNEUMONIA.**” is a bonafide and genuine research work carried out by me under the guidance of **Dr. VIDYASAGAR CR,MD**, Associate Professor, Department of General Medicine, SDUMC, Kolar.

Date:

Place: Kolar

**Dr. UPHAR GUPTA**

### **CERTIFICATE BY THE GUIDE**

This is to certify that this dissertation entitled “**COMPARISON OF VALIDITY OF SEVERITY SCORING SYSTEMS IN COMMUNITY ACQUIRED PNEUMONIA.**” Is a bonafide research work done by **Dr. UPHAR GUPTA** in partial fulfillment of the requirement for the degree of **Doctor Of Medicine In GENERAL MEDICINE**, SDUMC, Kolar.

Date :

Place : Kolar

**SIGNATURE OF THE GUIDE**

**Dr. VIDYASAGAR C.R. M.D.**

Associate Professor,

Department Of General Medicine

Sri Devaraj Urs Medical College, Tamaka,  
Kolar.

**ENDORSEMENT BY THE HOD,**  
**PRINCIPAL / HEAD OF THE INSTITUTION**

This is to certify that this dissertation entitled “**COMPARISON OF VALIDITY OF SEVERITY SCORING SYSTEMS IN COMMUNITY ACQUIRED PNEUMONIA**”. Is a bonafide research work done by **Dr. UPHAR GUPTA** in partial fulfillment of the requirement for the degree of **Doctor of Medicine In General Medicine**, SDUMC, Kolar.

**Dr. PRABHAKAR K M.D.**  
Professor & HOD  
Department of General Medicine,  
Sri Devaraj Urs Medical College,  
Tamaka, Kolar

**Dr. B.G. RANGANATH M.D.**  
Principal,  
Sri Devaraj Urs Medical College,  
Tamaka, Kolar

Date:  
Place:Kolar

Date:  
Place: Kolar

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND  
RESEARCH, TAMAKA, KOLAR, KARNATAKA**

**ETHICS COMMITTEE CERTIFICATE**

This is to certify that the Ethics committee of Sri Devaraj Urs Medical College & Research Center, Tamaka, Kolar has unanimously approved

***Dr. UPHAR GUPTA***

***Post-Graduate student in the subject of***

***DOCTOR OF GENERAL MEDICINE***

***at Sri Devaraj Urs Medical College, Kolar to take up the Dissertation  
work entitled***

**“COMPARISON OF VALIDITY OF SEVERITY SCORING  
SYSTEMS IN COMMUNITY ACQUIRED PNEUMONIA.”**

to be submitted to the

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND  
RESEARCH CENTER, TAMAKA, KOLAR, KARNATAKA,**

Date :  
Place : Kolar

**Member Secretary**  
Sri Devaraj Urs Medical College,  
Kolar-563101

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH**

**TAMAKA, KOLAR, KARNATAKA**

**COPY RIGHT**

**DECLARATION BY THE CANDIDATE**

I hereby declare that the Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purpose.

**Dr. UPHAR GUPTA**

Date :

Place : Kolar

## **ACKNOWLEDGEMENT**

*No academic work is single handedly accomplished. This work is no exception. Words fail me in expressing my heart felt and humble gratitude to my guide **Dr. VIDYASAGAR C.R.**, M.D., Professor, Department of General Medicine, for the guidance and encouragement all along in completing my study. His encouragement, sense of punctuality, research oriented approach, the painstaking effort to weed out errors and his affection during the entire course of study leaves me permanently indebted to him.*

*It is with deep sense of gratitude and respect, I have taken an opportunity to thank my teacher **Dr. PRABHAKAR K.**, M.D., Professor and HOD, Department of General Medicine, Sri Devaraj Urs Medical College, Tamaka, Kolar, for his constant inspiration and valuable guidance at various stages of preparation of this dissertation.*

*I am equally thankful to **Dr. B.N.RAGHAVENDRA PRASAD**, ex-HOD, Dept. Of General Medicine, without whose support the inception of this work would have been impossible and who has helped me through all stages of this work.*

*I express my deep sense of gratitude and humble thanks to **Dr.Lakshmiaiah V, Dr.P.N.Venkatarathnamma and Dr.Raveesha A**, Professors for their advice and constant encouragement throughout the present study.*

*I express my gratitude to **Dr. S.R.PRASAD**, professor & director of Post Graduate studies for his encouragement invaluable inputs for the study.*

*I would like to thank all my teachers Dr.Jayaram.N, Dr. Srinivasa.S.V , Dr.Harish Kumar, Dr.Pradeep Kumar, Dr. Reddy Prasad, Dr.Naveen.L, Dr.Santoshi Malkarnekar, Dr.Anto George, Dr.Naga Sumanth Reddy, Dr.Shankar, Dr. Niveditha, Dr. Prasanna and Dr. Vishwanath from the Department of General Medicine for their heartfelt support at all times.*

*I would like to acknowledge the opportune help and permission rendered by the Medical Director, Principal and Medical superintendent, SDUMC/RLJH, in conducting this study.*

*I humbly thank Dr.Srikanth Rammohan, Dr. Kiran BJ, Dr. Ujjwal Kumar and Dr. Anitha A my senior postgraduates for their help and guidance to complete my dissertation. I thank all my colleagues and junior post graduates for having rendered their cooperation during the study.*

*I am also thankful to all the Nursing Staff of the Central ICU and MICU, Technical Staff and non-teaching staff for their invaluable help without which this study would not have been possible.*

*I thank my parents Mrs. Archana Gupta and Dr. D.P.Gupta, my elder brother Dr. Utsav Gupta and my sister in law Dr. Supriti Gupta and my fiancé Dr. Hina Agrawal for their constant source of encouragement, and help during the period of my study.*

**Dr. UPHAR GUPTA**



## **ABSTRACT**

### **Comparison of Severity Scoring Systems in Predicting the Prognosis of Community Acquired Pneumonia.**

#### **BACKGROUND:**

Pneumonia has been considered a health problem for ages. Despite being the cause of significant morbidity and mortality. Delay in ICU admission of CAP patients has been shown to be associated with increased mortality. The two prominent tools for this purpose are the pneumonia severity index (PSI), developed in the USA after pneumonia outcome research trial (PORT), and the CURB-65 rule.

#### **OBJECTIVES OF THE STUDY:**

1. To compare the prognostic value (need for ICU admission and mortality) of the pneumonia severity index (PSI) and CURB-65 in the patients of community-acquired pneumonia.
2. To test the applicability of pneumonia severity index and CURB-65.

#### **MATERIALS AND METHODS:**

Eighty patients were assessed with both the scoring systems and total score for each patient for each scoring system was calculated. The patients' clinical outcome was also recorded within two weeks after admission. All the patients were assessed using PSI scoring and CURB65 scoring.

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Chi-square was used as test of significance. Independent t test was used as test of significance to identify the mean difference between two groups. p value <0.05 was considered as statistically significant.

#### **RESULTS:**

A significant association between Age, on ventilator support, on inotropes, ICU stay and Mortality was observed with increasing scores in both PSI and CURB-65 scoring systems in our study, with a high degree of sensitivity and specificity.

Whereas PSI score has higher prediction for ICU admission and Ventilator requirement in CAP patients, the CURB Score has higher prediction for Mortality in CAP patients. In our study in the ROC curve Area under the curve for ICU admitted patients, patients having received Ventilator Support and Mortality among CAP subjects was higher for PSI score than CURB-65. I.e. PSI score has higher Sensitivity, Specificity and Area under Curve (AUC) for Mortality for all the three aforementioned parameter in CAP patients.

#### **CONCLUSION:**

The comparison between mortality rates in different risk classes in our study and that of the previous studies showed that in all the studies mortality rates progressively increases with increasing risk scores in both PSI and CURB-65 risk classes. In predicting ICU admission, ventilatory support, inotropic support and Mortality both PSI and CURB65 has good specificity with PSI having a better sensitivity and specificity than CURB65.

**KEYWORDS:** PSI , CURB-65, Community Acquired Pneumonia

## **ABBREVIATIONS**

BTS	British Thoracic Society
CT	Computed Tomography
CAP	Community Acquired Pneumonia
CURB	Confusion, Urea, Creatinine, Blood Pressure
HCAP	Health Care–Associated Pneumonia
HAP	Hospital Acquired Pneumonia
ICU	Intensive Care Unit
IL	Interleukin
LRTI	Lower Respiratory Tract Infection
MRSA	Methicillin Resistant Staphylococcus Aureus
PSI	Pneumonia Severity Index
PCR	Polymerase chain reaction
rAST	revised American Thoracic Society
RSVs	Respiratory Syncytial Viruses
SCAP	Severe Community-Acquired Pneumonia
TNF	Tumor Necrosis Factor
VAP	Ventilator Associated Pneumonia

## **TABLE OF CONTENTS**

		<b>Page #</b>
<b>1.</b>	<b>INTRODUCTION</b>	<b>01</b>
<b>2.</b>	<b>OBJECTIVES OF THE STUDY</b>	<b>03</b>
<b>3.</b>	<b>REVIEW OF LITERATURE</b>	<b>04</b>
<b>4.</b>	<b>MATERIALS AND METHODS</b>	<b>39</b>
<b>5.</b>	<b>RESULTS</b>	<b>44</b>
<b>6.</b>	<b>DISCUSSION</b>	<b>70</b>
<b>7.</b>	<b>CONCLUSION</b>	<b>83</b>
<b>8</b>	<b>SUMMARY</b>	<b>85</b>
<b>9.</b>	<b>BIBLIOGRAPHY</b>	<b>86</b>
<b>10.</b>	<b>ANNEXURES</b>	<b>99</b>

## **LIST OF TABLES**

<b>TABLE NO</b>	<b>TABLES</b>	<b>PAGE NO</b>
1	Classifications of pneumonia.	06
2	Causative Agents of CAP - Bacterial	06
3	Causative Agents of CAP - Non-bacterial pneumonia	07
4	Host Defence Mechanism	09
5	Microbial Causes of Community-Acquired Pneumonia, by Site of Care	15
6	Investigation Protocol	18
7	CURB-65 criteria Scoring	27
8	Port Prediction Rule	29
9	Age Distribution of subjects	44
10	Gender distribution	46
11	Symptoms in Subjects	47
12	Comorbidities in Subjects	48
13	Personal History of subjects	49
14	Mean and SD of Various Parameters	50
15	Complications in Community Acquired Pneumonia cases	52
16	X- ray Findings of Lobe Involved in CAP subjects	53
17	Distribution of subjects according to ICU stay	56

18	Distribution of subjects according to Ventilator support	57
19	Distribution of subjects according to Mortality	58
20	Distribution of subjects according to Inotropes used	59
21	Distribution of PSI Score in CAP subjects	60
22	Distribution of CURB – 65 Score in CAP subjects	61
23	Association between CURB – 65 score with various parameters	62
24	Association between PSI score with various parameters	63
25	Sensitivity, Specificity, PPV, NPV and accuracy of CURB 65 for predicting Mortality	64
26	Sensitivity, Specificity, PPV, NPV and accuracy of PSI for predicting Mortality	65
27	Sensitivity, Specificity and Area under Curve (AUC) of PSI and CURB65 for Need for Admission to ICU	67
28	Sensitivity, Specificity and Area under Curve (AUC) of PSI and CURB65 for Need for ventilation.	68
29	Sensitivity, Specificity and Area under Curve (AUC) of PSI and CURB65 for Mortality	69
30	Comparison of Distribution of PSI	74
31	Comparison of Distribution of CURB-65	75
32	Comparison Of Mortality As Outcome By Psi	76
33	Comparison Of Mortality As Outcome By Curb65	77

## **LIST OF FIGURES**

<b>TABLE NO</b>	<b>FIGURES</b>	<b>PAGE NO</b>
1	Morbid anatomist's classification of Pneumonia	05
2	The Host Defense Mechanisms	11
3	Pathogenesis Of CAP	14
4	Gram Stain Depiction of Streptococcus Pneumoniae	20
5	Gram Stain Depiction of Haemophilus Pneumoniae	20
6	Chest Radiographs showing Consolidation	23
7	Inotropic and Ventilatory Support being given to a patient of CAP	41
8	Arterial Blood Gas Analyzer	41
9	Radiographic Depiction of CAP	42
10	Few Radiographs Depicting Various Patterns Of CAP	55

### **LIST OF GRAPHS**

<b>GRAPH NO</b>	<b>GRAPHS</b>	<b>PAGE NO</b>
1	Age distribution of subjects	45
2	Gender distribution of subjects	46
3	Symptoms in Subjects	47
4	Comorbidities in Subjects	48
5	Personal History of subjects	49
6	Vital signs	51
7	Metabolic Profile of subjects	51
8	Renal/ Electrolyte and Glycemic Profile	52
9	Complications in CAP	53
10	X- ray Findings of Lobe Involved	54
11	ICU admission in CAP subjects	56
12	Distribution of subjects according to Ventilator support	57
13	Distribution of subjects according to Mortality	58
14	Distribution of subjects according to Inotropes used	59
15	Distribution of PSI Score in CAP subjects	60
16	Distribution of CURB – 65 Score in CAP subjects	61
17	Sensitivity, Specificity, PPV, NPV and accuracy of CURB 65 for predicting Mortality	64
18	Sensitivity, Specificity, PPV, NPV and accuracy of PSI for predicting Mortality	65



19	ROC Curve - Need for Admission to ICU	66
20	ROC Curve - Need for Need For Ventilatory Support	67
21	ROC Curve – Comparison of Mortality	68
22	Inter - Study variation of Sex Distribution	71
23	Distribution of Cases - PSI Score	74
24	Distribution of Cases – CURB-65 Score	75
25	Comparison Of Mortality As Outcome By Psi Score	77
26	Comparison Of Mortality As Outcome By Curb65 Score	78
27	Comparison Of Prediction of Mortality in the highest Class Of Scoring System	87

## **INTRODUCTION**

In 1901 William Osler described pneumonia as the "captain of the men of death." He described the condition as 'the friend of the aged', allowing them a merciful release from 'those cold gradations of decay that make the last state of all so distressing' <sup>1</sup>. Pneumonia has been considered a health problem for ages. The outlook towards the disease has changed drastically with the availability of antibiotics and now cure is considered the rule and death, the exception.

Whereas pneumonia in the elderly is frequently a terminal event in a patient disabled or dying as a result of some other incurable disease, this is clearly not usually the case in younger previously healthy patient groups, such as military recruits or students, in whom mortality is low.

Pneumonia has been considered a health problem for ages. Despite being the cause of significant morbidity and mortality, Pneumonia is often misdiagnosed, mistreated, and underestimated.

The pneumonia is typically classified as<sup>2</sup>

1. Community-acquired,
2. Hospital-acquired, or Healthcare-associated.

Community-acquired pneumonia (CAP) is a major cause of morbidity and mortality worldwide. Community-acquired pneumonia (CAP) is a disease in which individuals who have not recently been hospitalized develop an infection of the lungs (pneumonia). Lower respiratory tract infections (LRTIs), including CAP, were ranked third in a list of the 30 leading causes of death worldwide in 1990. Mortality rates are low (< 2%) in CAP patients treated as outpatients, but are higher (5 to 20%) among patients hospitalized for CAP, and are highest (up to 50%) in patients admitted to the intensive care<sup>3</sup>.

The importance CAP is increasing economically since it is considered the leading cause of absence to jobs, incapacity and activity restriction in developing countries.

The condition imposes a heavy burden on the healthcare system in terms of its high cost both for diagnosing and treating the condition as well as for the hospital and ICU stay.<sup>4</sup> This heavy cost points out the importance of predicting the need for hospitalization as well as the outcome of these patients. Prognostic scoring systems for CAP have been developed to address these issues. The two prominent tools for this purpose are the pneumonia severity index (PSI), developed in the USA after pneumonia outcome research trial (PORT), and the CURB-65 rule developed in the U.K. as “confusion, elevated blood urea nitrogen, elevated respiratory rate, low systolic or diastolic blood pressure (BP), and age over 65 years (CURB-65)” rule.<sup>5, 6</sup> The two scoring approaches are viewed as being complementary, as each has different strengths and weaknesses.

## **OBJECTIVES**

1. To compare the prognostic value (need for ICU admission and mortality) of the pneumonia severity index and CURB-65 in the patients of community-acquired pneumonia.
2. To test the applicability of pneumonia severity index and CURB-65 in the patients of community-acquired pneumonia.

## **REVIEW OF LITERATURE**

### **Definition**

Pneumonia is defined as an acute inflammation of the pulmonary parenchyma that can be caused by various infective and non-infective agents, presenting with physical and radiological features compatible with pulmonary consolidation of a part or parts of one or both lungs<sup>7</sup>

When the word 'pneumonia' is used in medical practice, it almost always refers to a syndrome caused by acute infection, usually bacterial, characterized by clinical and/or radiographic signs of consolidation of a part or parts of one or both lungs. Pneumonitis is occasionally used as a synonym for pneumonia, particularly when inflammation of the lung has resulted from a non-infectious cause, such as chemical or radiation injury.

About 15 million children worldwide die each year as a consequence of acute respiratory infections, one-third of them from pneumonia, and 96% of these deaths occur in developing countries<sup>8,9</sup>.

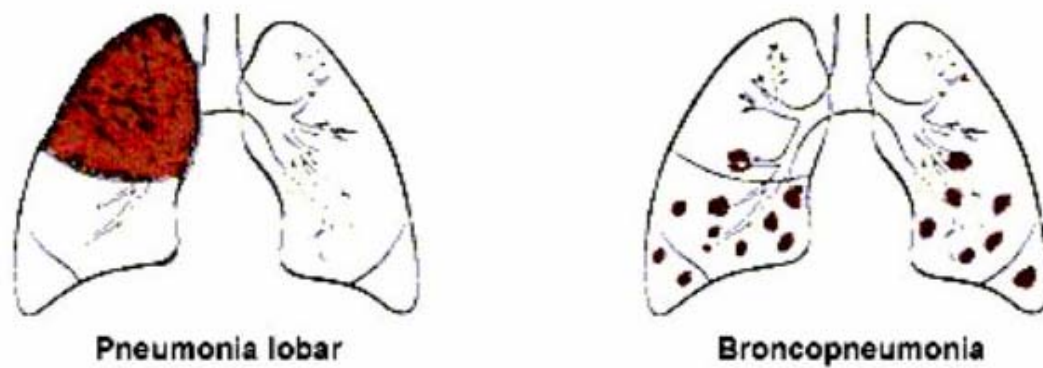
Certain sections of the community, such as drug abusers, are susceptible to pneumonia. This may arise due to 'seeding' of the lung by staphylococci or other organisms from right-sided infective endocarditis<sup>10</sup>, or as a result of the aspiration of oropharyngeal contents while in a stuporose state, which may result in a predominantly anaerobic or Gram-negative pneumonia

The death rates from pneumonia may be influenced by seasonal factors, being greater in the cold winter months than in the summer. This difference is more evident in lower socioeconomic groups and is unaccounted for by influenza epidemics alone<sup>11</sup>. It is

possible that greater overcrowding and poorer ventilation in cold weather may be factors enabling the spread of infection. In contrast, pneumonia due to *Legionella pneumophila* tends to occur more commonly in the warmer months.

### **Classification**

No categorization of pneumonia is entirely satisfactory (Table 1) but for descriptive purposes the classification should be both anatomical (the terms used communicate the extent and distribution of the process in the lung or lungs) and causal (the responsible microorganism is named).



**Fig 1: Morbid anatomist's classification<sup>5</sup>**

**Table 1: Classifications of pneumonia.<sup>7</sup>**

<b>Morbid anatomist's classification</b>	<b>Empiricist's classification</b>	<b>Behaviourist's classification</b>
Lobar pneumonia	Community-acquired pneumonia	Easy pneumonia (responds to initial treatment)
Segmental pneumonia	Hospital-acquired (nosocomial) pneumonia	Difficult pneumonia (fails to do so)
Sub segmental pneumonia	Aspiration pneumonia	
Bronchopneumonia	Immunocompromised host pneumonia	

**Table 2: Causative Agents of CAP – Bacterial<sup>7</sup>**

**Pneumococcal pneumonia**

*Streptococcus pneumoniae*

**Atypical pneumonia**

*Legionella* spp. (legionnaires')

*Mycoplasma pneumoniae*

*Chlamydia* spp.

*Coxiella burnetii* (Q fever).

**Staphylococcal pneumonia**

*Staphylococcus aureus*

**Gram-negative enteric pneumonia**

*Klebsiella* spp.

*Pseudomonas aeruginosa*

*Escherichia coli*

*Enterobacter* spp.

*Serratia* spp.

***Haemophilus influenzae* pneumonia**

***Moraxella catarrhalis* pneumonia**

**Anaerobic pneumonia (mixed flora)**

*Bacteroides* spp.

*Fusobacterium* spp.

*Peptococcus* spp.

*Peptostreptococcus* spp.

**Mycobacterial pneumonia**

*Mycobacterium tuberculosis*.

**Table 3 Causative Agents of CAP - Non-bacterial pneumonia<sup>7</sup>**

<b>Sl No</b>	<b>Causative Agents of CAP</b>
1	Viral pneumonia
2	Influenza
3	Measles
4	Adenoviruses
5	Varicella
6	Cytomegalovirus
7	Respiratory syncytial virus
8	Parainfluenza virus
9	Coronaviruses
10	Coxsackie virus
11	Rhinoviruses
12	Epstein–Barr virus
13	Herpes simplex virus
14	Hantavirus, etc
15	Bacteria-like and rickettsia-like pneumonia
16	Fungal and actinomycotic pneumonia
17	Parasitic pneumonia
18	Chemical pneumonia, e.g. lipoid
19	Physical pneumonia, e.g. ionizing radiation



The causal organism can only be guessed at when the patient is first seen and it is useful in this respect to classify the case as one of either community-acquired or hospital acquired (nosocomial) pneumonia. These two groups are not mutually exclusive for particular pathogens but the spectrum of infecting organisms in the two groups does vary, partly because the hospital population has selected disproportionate numbers of elderly patients and those whose bacterial flora has been modified as a result of their stay as well as those whose immune defences are compromised by severe underlying disease or suppressed by drugs. Nosocomial pneumonia is a particular problem in postoperative patients and in those treated in intensive care units, the latter group being highly susceptible to lower respiratory tract infection<sup>12-14</sup>.

The different lung pathogens found in hospitals result from the alteration in bacterial flora caused by the use of antibiotics and also often from the instrumentation or intubation of the upper airways of patients, which provides the organisms with easy access to the lungs. Such hospital-acquired infections are more frequently due to aerobic Gram negative bacilli and *Staphylococcus aureus* (increasingly methicillin resistant) compared with those acquired in the community<sup>15-17</sup>.

Community acquired pneumonia (CAP) is a common disorder with an incidence of about 20% to 30% in developing countries compared to an incidence of 3% to 4 % in developed countries.<sup>18,19</sup> In children, most deaths (over two million a year) occur in newborn period. According to a World Health Organization estimate, one in three newborn deaths are from pneumonia. Mortality decreases with age until late adulthood, with the elderly at risk for CAP and its associated mortality<sup>20</sup>.

Community acquired pneumonia is defined as an acute illness acquired in the community with symptoms suggestive of LRTI (lower respiratory tract infection), together with presence of a chest radiograph of intra pulmonary shadowing which is likely to be new and has no clear alternative cause with symptoms occurring outside of the hospital or within 48 hours of hospital admission in a patient not residing in a long-term care facility.<sup>7, 21</sup>

### **Lung defence mechanisms**

The host mechanisms has been classified based upon the location<sup>22</sup>:-

**Table 4 : Host Defence Mechanism**

<b>Location</b>	<b>Upper Airways</b>
	Nasal hair
	Turbinates
	Mucociliary apparatus
	Immunoglobulin A (IgA) secretion
<b>Oropharynx</b>	Saliva
	Sloughing of epithelial cells
	Local complement production
	Interference from resident flora
	Conducting Airways
<b>Trachea, bronchi</b>	Cough, epiglottic reflexes
	Sharp-angled branching of airways
	Mucociliary apparatus
	Immunoglobulin production (IgG, IgM, IgA)
	<b>Lower Respiratory Tract</b>
<b>Terminal airways, alveoli</b>	Alveolar lining fluid (surfactant, Ig, complement, fibronectin)
	Cytokines (interleukin 1, tumour necrosis factor)
	Alveolar macrophages
	Polymorphonuclear leukocytes
	Cell-mediated immunity

The host mechanisms can also be classified based upon the time and mechanism of operation<sup>22</sup>:-

**A. Mechanisms that operate at birth i.e, innate immunity**

1. In the nonimmune lung, removal of microbial organisms depends on entrapment in the mucous blanket and removal via the mucociliary elevator,
2. Phagocytosis by alveolar macrophages that can kill and degrade organisms and remove them from the airspaces by migrating onto the mucociliary elevator, or
3. phagocytosis and killing by neutrophils recruited by macrophage factors.
4. Serum complement may enter the alveoli and be activated by the alternative pathway to provide the opsonin C3b that enhances phagocytosis. (5) Organisms, including those ingested by phagocytes, may reach the draining lymph nodes to initiate immune responses.

**B. Additional mechanisms operate after development of adaptive immunity**

1. Secreted IgA can block attachment of the microorganism to epithelium in the upper respiratory tract.
2. In the lower respiratory tract, serum antibodies (IgM, IgG) are present in the alveolar lining fluid. They activate complement more efficiently by the classic pathway, yielding C3b. In addition, IgG is opsonic.
3. The accumulation of immune T cells is important for controlling infections by viruses and other intracellular microorganisms. PMN, polymorphonuclear cells.

- Defect in host defense mechanism
- Overwhelming inoculum



Philadelphia, PA: Saunders/Elsevier, 2007)

Pneumonia is predisposed by any condition that

- i. Reduces or suppresses the cough,
- ii. Impairs mucociliary activity,
- iii. Reduces the effective phagocytic activity of alveolar macrophages and neutrophils,

- iv. Impairs immunoglobulin production. Potential pathogens reach the lung to cause pneumonia chiefly by microaspiration of secretions containing oropharyngeal flora, but also by overt aspiration, by inhalation from the environment, from a nebulizer or anaesthetic circuit and by blood spread.<sup>23</sup>

#### **Aspiration and microaspiration:**

- It occurs following surgery, general anesthesia, tracheostomy, or the passage of endotracheal or nasogastric tube<sup>24</sup>.
- Organisms like *S. pneumoniae*, *H. influenza*, *S. aureus*, anaerobic bacteria may exist commensally in oropharynx<sup>25-26</sup>.
- Favourable conditions for infection may also be provided by chemical injury to the lungs resulting from overt gastric acid aspiration or by pulmonary oedema, and alcoholism is an important predisposing factor<sup>27</sup>.

#### **Inhalation**

The inhalation of microbes contained in small particle aerosols is thought to be important in the transmission of viral infections and also in *Legionella pneumonia*. The inhalation of infected particles from animals may be responsible for psittacosis and *Coxiella pneumonia* (Q fever).

Pathogens may also be introduced to the lower respiratory tract by contaminated nebulizer circuits or other respiratory equipment, this route being avoidable by proper preventive measures<sup>28, 29</sup>.

**Colonization:**

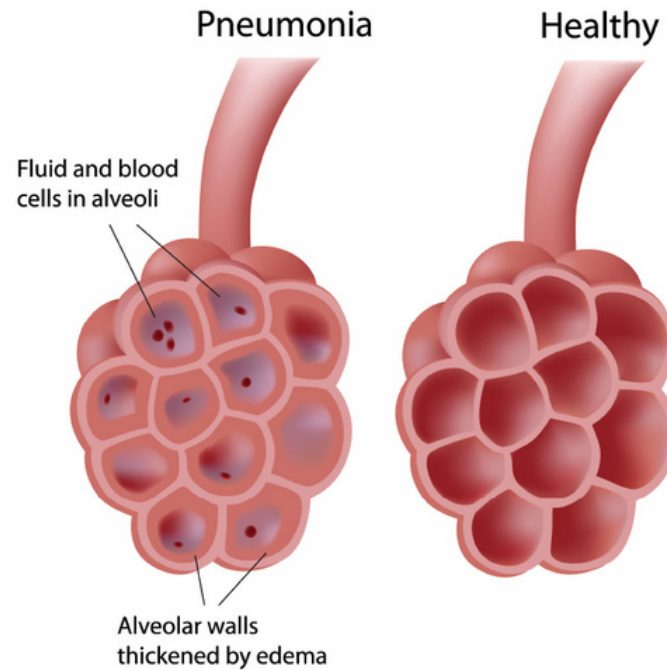
- It occurs in patients with pre-existing lung diseases like chronic bronchitis, emphysema, bronchiectasis, and cystic fibrosis<sup>30</sup>.

**Blood spread:**

- Hematogenous spread- with gram negative and staphylococcal bacteremia<sup>31-33</sup>.
- Patients with intravenous cannulae, chronic hemodialysis are more susceptible<sup>34</sup>.

**PATHOLOGY**<sup>21</sup>**4 phases:**

1. Edema: proteinaceous exudate and bacteria in the alveoli.
2. Red hepatization: Erythrocytes are present in the cellular intraalveolar exudate. Bacteria are occasionally seen.
3. Gray hepatization:
  - No new erythrocytes are extravasating.
  - Neutrophil is the predominant cell, fibrin deposition is abundant, and bacteria have disappeared.
  - Corresponds with successful containment of the infection and improvement in gas exchange.
4. Resolution: the macrophage is the dominant cell, and the debris of neutrophils, bacteria, and fibrin has been cleared.



**Fig 3: Pathogenesis Of CAP**

(Adapted From Kumar, Vinay, and Stanley L. 1915- Robbins. *Robbins Basic Pathology*. 8th ed.

Philadelphia, PA: Saunders/Elsevier, 2007.)

## ETIOLOGY

The extensive list of potential etiologic agents in CAP includes bacteria, fungi, viruses, and protozoa<sup>8, 26</sup>.

Most cases of CAP are caused by relatively few pathogens & include,

1. *S.pneumoniae*,
2. *Haemophilus influenzae*,
3. *Moraxella catarrhalis*
4. *S.aureus*
5. Gram-negative bacilli such as *Klebsiella pneumoniae* ,*Pseudomonas aeruginosa*.

The "Atypical" organisms include

1. Mycoplasma pneumoniae,
2. Chlamydo phil pneumoniae, and
3. Legionella spp.
4. Respiratory viruses such as Influenza viruses, Adenoviruses, and Respiratory Syncytial Viruses (RSVs)

Outpatients	Non-ICU	ICU
Streptococcus pneumoniae	S. pneumoniae	S. pneumoniae
Mycoplasma pneumoniae	M. pneumoniae	Staphylococcus aureus
Haemophilus influenzae	Chlamydophila pneumoniae	Legionella spp.
C. pneumoniae	H. influenzae	Gram-negative bacilli
Respiratory viruses	Legionella spp.	H. influenzae

**Table 5 -Microbial Causes of Community-Acquired Pneumonia, by Site of Care<sup>9</sup>**

Anaerobes play a significant role only when an episode of aspiration has occurred days to weeks before onset of pneumonia. The combination of an unprotected airway (e.g., in patients with alcohol or drug overdose or a seizure disorder) and significant gingivitis constitutes the major risk factor. Anaerobic pneumonias are often complicated by abscess formation and significant empyema or par pneumonic effusions<sup>38</sup>.



## **HIGH-RISK GROUPS<sup>39</sup>**

- Age older than 65 years
- Smoke cigarettes
- malnourished due to health conditions
- underlying lung disease, including cystic fibrosis, asthma, or chronic obstructive pulmonary disease (emphysema)
- other underlying medical problems, including diabetes or heart disease
- Have a weakened immune system due to HIV, organ transplant, chemotherapy, or chronic steroid use
- difficulty coughing due to stroke, sedating drugs or alcohol, or limited mobility
- Have had a recent viral upper respiratory tract infection including influenza

## **DIAGNOSIS**

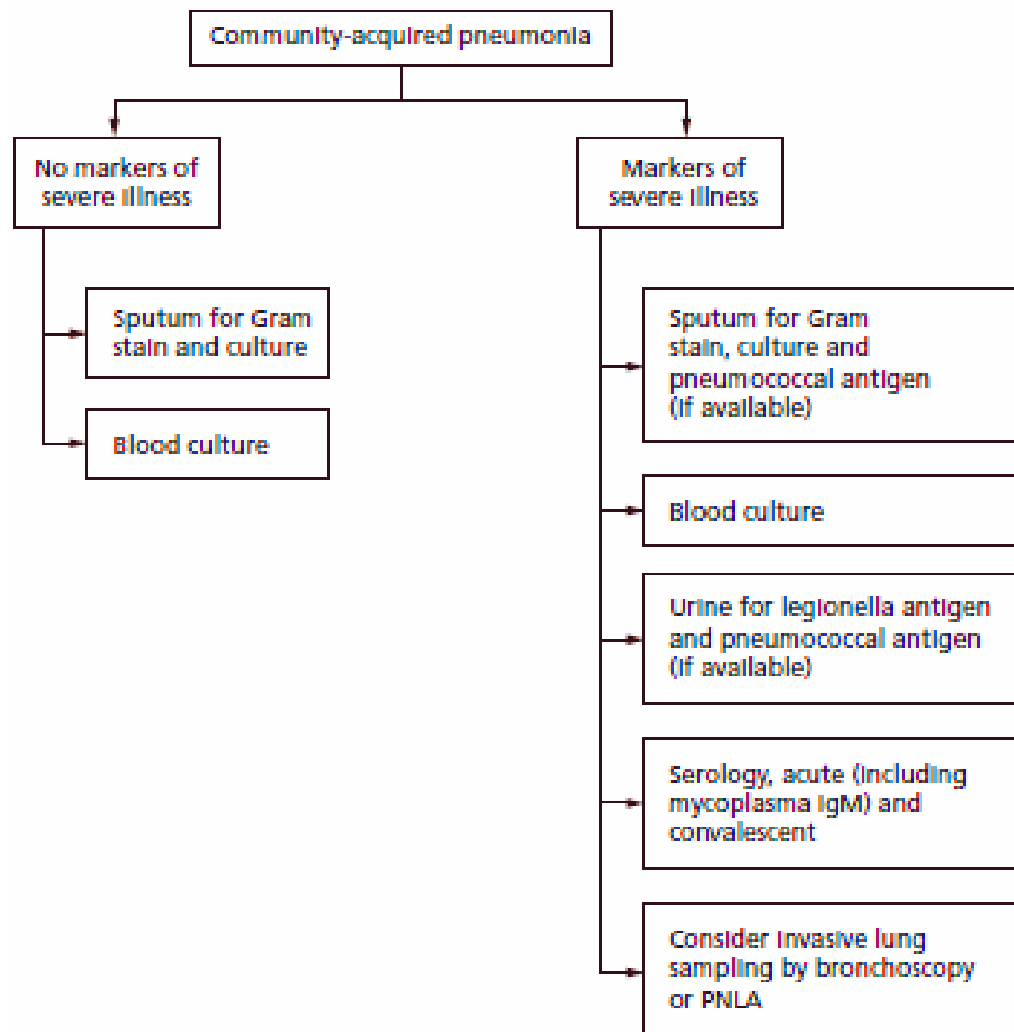
Recommended Tests for more severely ill and hospitalized patients with community-acquired pneumonia<sup>40</sup>:-

1. Chest Radiography
2. Sputum culture and sensitivity
3. Blood culture
4. Blood gas
5. Routine haematology and biochemistry
6. Rapid urine antigen testing for *S. pneumonia* and *Legionella* spp. serogroup 1
7. Thoracocentesis for pl. effusions

## Markers of severity in pneumonia at initial assessment

The lengths to which the clinician is prepared to investigate the microbiological cause of a case of pneumonia is likely to be determined by the severity of the illness at presentation<sup>40</sup>.

- Altered mental state/confusion
- Tachypnea ~30 breaths/min
- Hypotension,(SBP <90 mmHg, DBP < 60mmHg or need for vasopressors)
- Pao<sub>2</sub> <60mmHg
- PacO<sub>2</sub> >6.5 kPa (50mmHg) or consideration of the need for mechanical ventilation
- Chest radiograph shows more than one lobe involved or rapid progression<sup>41</sup>
- Evidence of renal insufficiency (serum urea~7mmol/L' low urine output < 20 mL/h )
- Need for admission to intensive care unit



**Table 6 – Investigation Protocol<sup>7</sup>**

### **Etiologic Diagnosis<sup>38,42</sup>**

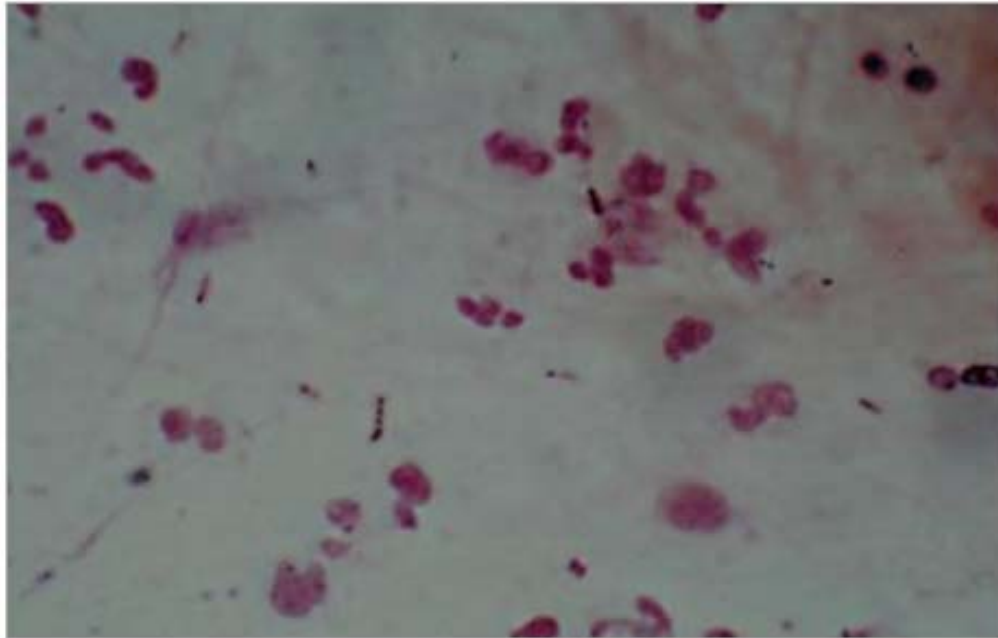
The etiology of pneumonia usually cannot be determined on the basis of clinical presentation; instead, the physician must rely upon the laboratory, no data exist to show that treatment directed at a specific pathogen is statistically superior to empirical therapy.

## **Gram's Stain and Culture of Sputum** <sup>43</sup>

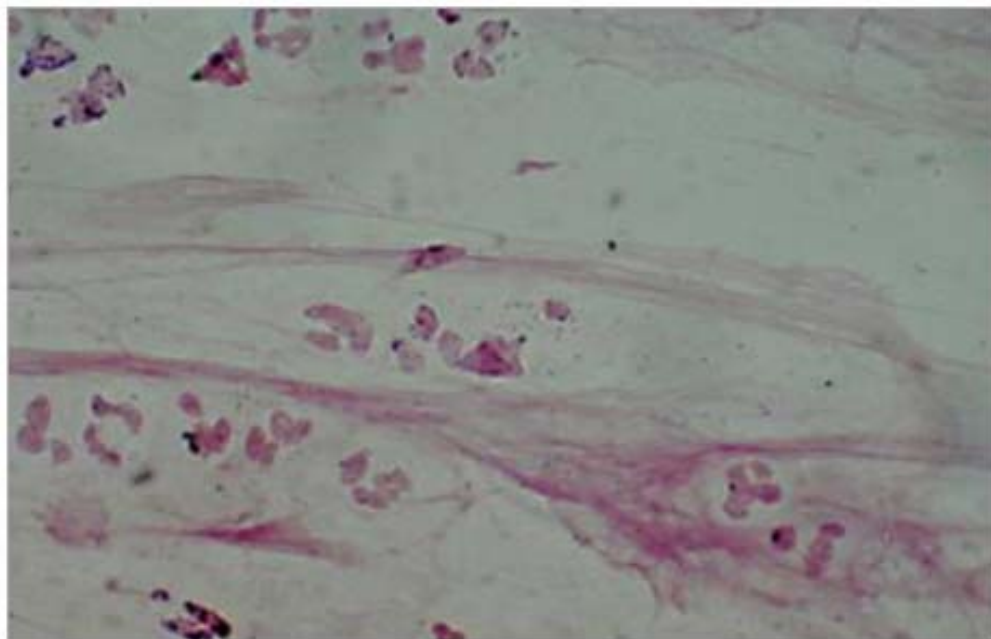
The main purpose of the sputum Gram's stain is to ensure that a sample is suitable for culture.

However, Gram's staining may also help to identify certain pathogens (e.g., *S. pneumoniae*, *S. aureus*, and gram-negative bacteria) by their characteristic appearance. To be adequate for culture, a sputum sample must have >25 neutrophils and <10 squamous epithelial cells per low power field<sup>44</sup>. The sensitivity and specificity of the sputum Gram's stain and culture are highly variable<sup>45</sup>; even in cases of proven bacteremic pneumococcal pneumonia, the yield of positive cultures from sputum samples is < 50%. Some patients may not be able to produce an appropriate expectorated sputum sample. Others may already have started a course of antibiotics, which can interfere with results, at the time a sample is obtained.

The greatest benefit of staining and culturing respiratory secretions is to alert the physician of unsuspected and/or resistant pathogens and to permit appropriate modification of therapy. Other stains and cultures may be useful as well. For suspected tuberculosis or fungal infection, specific stains are available. Cultures of pleural fluid obtained from effusions >1 cm in height on a lateral decubitus chest radiograph may also be helpful.



**Figures 4 : Gram Stain Showing Streptococcus pneumoniae**



**Figures 5: Gram Stain Depiction of Haemophilus Pneumoniae**

### **Blood Cultures<sup>45</sup>**

The yield from blood cultures, even those obtained before antibiotic therapy, is disappointingly low. Only 5–14% of cultures of blood from patients hospitalized with CAP are positive, and the most frequently isolated pathogen is *S. pneumoniae*. Since recommended empirical regimens all provide pneumococcal coverage, a blood culture positive for this pathogen has little, if any, effect on clinical outcome. However, susceptibility data may allow a switch from a broader-spectrum regimen to penicillin in appropriate cases. Because of the low yield and the lack of significant impact on outcome, blood cultures are no longer considered must for all hospitalized CAP patients. Certain high-risk patients—including those with neutropenia secondary to pneumonia, asplenia, or complement deficiencies; chronic liver disease; or severe CAP—should have blood cultured.

### **Antigen Tests<sup>44</sup>**

Two commercially available tests detect pneumococcal and certain *Legionella* antigens in urine. The test for *Legionella pneumophila* detects only serogroup 1, but this serogroup accounts for most community-acquired cases of Legionnaires' disease. The sensitivity and specificity of the *Legionella* urine antigen test are as high as 90% and 99%, respectively. The pneumococcal urine antigen test is also quite sensitive and specific (80% and >90%, respectively). Both tests can detect antigen even after the initiation of appropriate antibiotic therapy and after weeks of illness. Other antigen tests include a rapid test for influenza virus and direct fluorescent antibody tests for influenza virus and RSV.

### **Polymerase Chain Reaction<sup>46</sup>**

Polymerase chain reaction (PCR) tests are available for a number of pathogens, including *L. pneumophila* and mycobacteria. However, the use of these PCR assays is generally limited to research studies.

### **Serology<sup>42</sup>**

A fourfold rise in specific IgM antibody titer between acute- and convalescent-phase serum samples is generally considered diagnostic of infection with the pathogen in question.

### **Chest radiography**

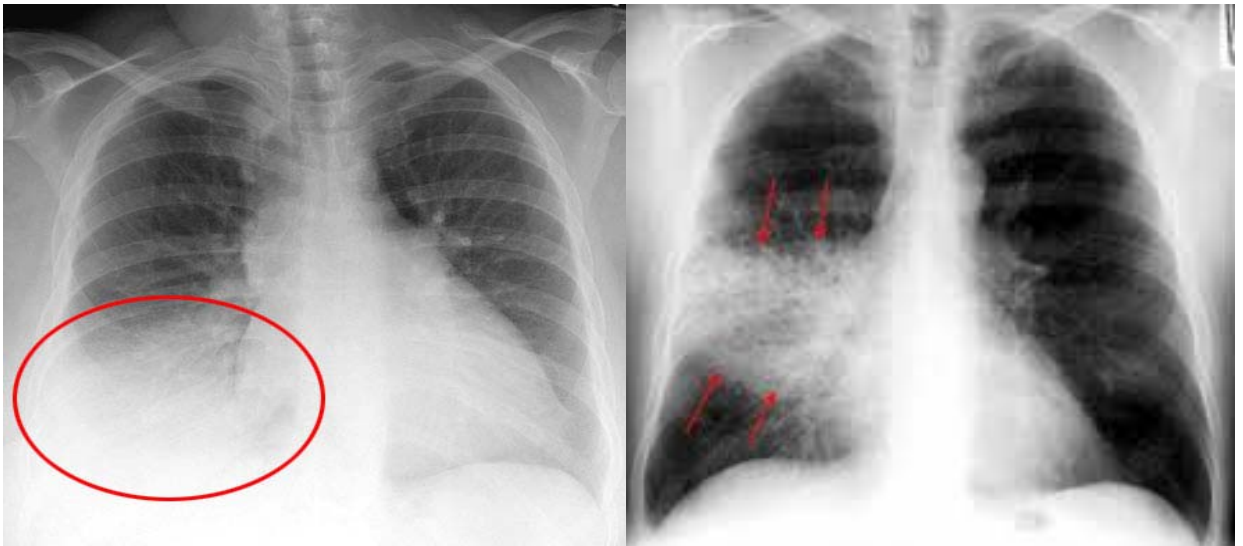
Chest radiographs should be obtained in all patients with suspected community-acquired pneumonia (CAP) to exclude conditions that mimic CAP and to confirm the presence of an infiltrate compatible with the presentation of CAP. Patients presenting very early with CAP may have negative findings on chest radiography. In these patients, repeat chest radiography within 24 hours.

Chest radiography assists with the differentiation of viral pneumonias from non-viral pneumonias.

- Viral pneumonias display few or no infiltrates on chest radiography, but when infiltrates are present, they are almost always bilateral, perihilar, symmetric, and interstitial

- Bacterial pneumonias have a predominantly focal segmental or lobar distribution. In contrast, typical or atypical pathogens produce a lobar or segmental pattern on chest radiography, with or without consolidation or pleural effusion.
- Pneumatoceles suggest infection with *S. aureus*, and an upper-lobe cavitating lesion suggests tuberculosis.
- Radiographic response to treatment usually lags well behind clinical improvement and pneumococcal pneumonia may take 6 weeks to clear on the chest film.

Persistent, recurrent or worsening shadowing may indicate either inappropriate treatment or bronchial obstruction by a foreign body or, more commonly, tumour particularly in patients over the age of 60 years <sup>48</sup>. Here are few examples non homogenous opacifications suggestive of community acquired pneumonia:-



**Radiographic Depiction of CAP**



## **Biomarkers**

These are typically performed if, based on extrapulmonary findings, atypical community-acquired pneumonia (CAP) is suspected.

Workup should include serum transaminase levels, serum phosphorous levels, urinalysis, ferritin levels, creatine phosphokinase (CPK) levels, C-reactive protein (CRP) levels, and cold agglutinin titers.

- The two currently in use are C-reactive protein(CRP) and procalcitonin (PCT).
- CRP may be of use in the identification of worsening disease or treatment failure.
- PCT may play a role in determining the need for antibacterial therapy.
- Hypophosphataemia may also occur <sup>49</sup>.
- Hyponatraemia due to inappropriate antidiuretic hormone (ADH) secretion may occur in any pneumonia and is notably more common in Legionella infection.

## **Treatment**

### **Site of Care**

The mortality of patients with severe Community Acquired Pneumonia(CAP) requiring admission to an intensive care unit (ICU) is high. This is likely to be particularly evident in a developing country like ours where availability of ICU beds is limited and only critically ill patients in need of assisted ventilation are admitted.<sup>50,51</sup> Concurrently, delay in ICU admission of CAP patients has been shown to be associated with increased mortality.<sup>52-54</sup> Certain patients clearly can be managed at home, and others clearly require treatment in the hospital, but the choice is sometimes difficult. Tools that objectively assess the risk of adverse outcomes, including severe illness and death, help to identify patients who will benefit from hospital care.

Patients with severe CAP, including children who require invasive ventilation via a non-permanent artificial airway, require admission to an intensive care unit (ICU). Oxygen and/or ventilatory support may be required.<sup>55</sup>

In the initial management of patients with suspected CAP the clinician is faced with diagnostic and prognostic challenges, each challenge corresponding to a specific management decision. This emphasises the importance of prompt, accurate diagnosis and severity of illness which corresponds to decisions regarding the intensity of management. The decision regarding the most appropriate site of care, including whether admission to hospital is warranted, is the first and single most important decision in the overall management of CAP. It has consequences both for the level of treatment received by the patient as well as the overall costs of treatment.<sup>57</sup>

An unchanged mortality of 4% to 21%<sup>58,59</sup> in-hospital treated CAP has renewed the interest in studying prognostic factors associated with fatal outcome.

The first landmark study to prognosticate patients of CAP was conducted by the Research Committee of the BTS in 1982.<sup>26</sup> In this study<sup>60</sup> comprising of 453 adults in 25 British hospitals, patients had a 21-fold increased risk of death if they had two of the following at admission: respiratory rate >30/min, diastolic BP <60mmHg, urea >7mmol/L. On the basis of these findings, BTS1 rule was constructed by selecting three factors, which were highly associated with death at admission, namely, respiratory rate >30/min at admission; diastolic blood pressure <60mmHg, and blood urea level >7mmol/L. This rule yielded the highest value among any of the rules tested in the Youden index, a statistic combining sensitivity and specificity for selection of an optimal rule, assuming equal importance of sensitivity and specificity.<sup>61</sup> When the first rule was

modified to use only three most predictive features ('confusion' replacing 'urea>7mmol/L), immediate application was possible with this second rule referred to as BTS2 rule. This modified rule had the highest overall accuracy (93%) and the highest specificity (94%) of any rule tested, but correctly identified only 39% of the patients who died; a positive rule was associated with a relative risk of death of 10.2.

These two rules were compared with a more complicated one suggested by Macfarlane,<sup>62</sup> which required at least three of the following factors: (i) confusion on examination, (ii) white blood cell count >10x10<sup>9</sup>/L or lymphocytes >1x10<sup>9</sup>/L; (iii) arterial oxygen tension (PaO<sub>2</sub>) 6.6KPa; and (iv) blood urea level >7mmol/L. It showed an overall accuracy of 87%, but identified only 50% of the patients who died, and was associated with a relative risk of death of 6.4. Neill et al<sup>37</sup> derived a modified BTS rule (mBTSr) in which severe CAP was suggested by the presence of two or more of: (i) confusion, (ii) respiratory rate >30/ minute, (iii) diastolic BP <60mmHg; and (iv) blood urea >7mmol/L at the time of admission.

Subsequently, CURB criteria (confusion, urea, respiratory rate and blood pressure) were developed which were similar to mBTSr, but systolic BP <90mmHg was added (either systolic BP <90mmHg or diastolic BP <60mmHg scores 1). Authors<sup>30</sup> also suggested CURB-65 where an age >65 years was given additional score of 1, making a total score of 5.

A major breakthrough was achieved only after the transformation of these rules into a risk score, which resulted from adding one point for each of these parameters (CURB or for patients aged >65 years CURB-65) by Lim and co-workers.<sup>6,63</sup>

The scoring system consists of a six-point score determined at the time of initial presentation. CURB 65 is a modification of the British Thoracic Society (BTS) rule in assessing pneumonia mortality risk. Developed by Lim et al., the CURB 65 evaluates the risk of mortality in CAP by assigning a score of 1 for each of the five parameters i.e. Presence of confusion (C) , Blood urea nitrogen (U) >7 mmol/L , Respiratory rate (R)  $\geq 30/\text{min}$  , Blood pressure (B) <90 mmHg systolic or  $\leq 60$  mmHg diastolic. The patients are assigned classes based on total score i.e. scores 0-1 as class I, scores 2-3 as class II and scores 4-5 as class III.<sup>64</sup>

---

**Confusion**

**Blood urea >7 mmol/L at the time of admission.**

**Respiratory Rate of  $\geq 30/\text{minute}$**

**Systolic BP  $\leq 90$  mmHg or diastolic BP  $\leq 60$  mmHg**

**Age  $\geq 65$  years**

---

**A score of 1 is given for presence of each of the variables**

**BP=Blood pressure**

---

**Table 7- CURB-65 criteria Scoring**

Pneumonia Severity Index is designed to predict CAP mortality and identify patients who are at a low risk of death and thus provide outpatient care for this cohort. It is a mortality prediction tool that was first introduced by Fine et al. in 1997. The rule was validated with 1991 data on 38,039 inpatients and with data on 2287 inpatients and outpatients in the Pneumonia Patient Outcomes Research Team (PORT) cohort study.<sup>56</sup> The original role of the PSI was to identify those patients at a low risk of

mortality who, therefore, could safely be treated as out-patients. The PSI was subsequently confirmed to make valid predictions of mortality by several authors, although in some reports mortality rates were somewhat lower in the highest risk group.<sup>65-67</sup>

The rule stratifies patients into five classes of risk for death within 30 days of presentation. The lowest risk class (risk class I) comprises patients who are younger than 50 years of age, have none of the five important coexisting illnesses and have normal mental status and normal or only mildly abnormal vital signs at presentation. Assignment to the remaining risk classes depends on the presence or absence of a set of medical history, physical examination, and laboratory findings. Finally, the PSI was also shown to predict long-term outcomes of CAP.<sup>68</sup> A major limitation of the PSI is the unbalanced impact of age on the score, resulting in a potential underestimation of severe pneumonia, particularly in younger otherwise healthy individuals.<sup>65</sup> Nevertheless, the PSI is currently recommended as a tool of severity assessment in the Infectious Diseases Society of America (IDSA) guidelines.<sup>69,70</sup>

The PSI score is assigned after assessing the patient with the PORT prediction rule. The scores for each of the point are assigned and total score is calculated. Based on the total points, the approach for site-of care is made. Patients with total point less than 50 are in class I, between 51-90 in class II, between 71-90 in class III, 91-130 in class IV and more than 130 in class V.

Patient characteristics	Points
Age (years)	1 point per year subtract 10 points for female
Coexisting illness	
Neoplastic disease	30
Liver disease	20
Congestive heart failure, cardiovascular disease, renal disease	10
Physical examination findings	
Altered mental status	
Respiratory rate $\geq 30$ breaths/min, systolic blood pressure $< 90$ mmHg, confusion	20
Temperature $< 35^{\circ}\text{C}$ ( $95^{\circ}\text{F}$ ), $> 40^{\circ}\text{C}$ ( $104^{\circ}\text{F}$ )	15
Pulse $> 125$ beats/min	10
Lab findings	
Blood urea nitrogen $> 20$ mg/dL; Sodium $< 130$ mmol/L	20
Glucose $> 250$ mg/dL, Hematocrit $< 30\%$	10
Partial pressure of arterial oxygen $< 60$ mmHg	10
Pleural effusion	10

**TABLE 8 : PORT PREDICTION RULE<sup>5</sup>**

Risk	Class	Score
Low	I	$< 51$
Low	II	51 - 70
Low	III	71 - 90
Medium	IV	90 - 130
High	V	$> 130$

## **Initial Antibiotic Management**

Initial therapy is usually empirical and is designed to cover the most likely pathogens . In all cases, antibiotic treatment should be initiated as expeditiously as possible<sup>71</sup>.

### **Empirical Antibiotic Treatment of Community-Acquired Pneumonia**

#### **OUTPATIENT MANAGEMENT**

1. Previously healthy patients without comorbidities and no use of antimicrobials within the prior 3 months A macrolide (preferred) (e.g., clarithromycin, extended release, 1000 mg orally each day for at least 5 days, or azithromycin, 500 mg orally on day 1, followed by 250 mg orally each day on days 2-5) Doxycycline, 100 mg orally twice daily for at least 5 days
2. Presence of comorbidities such as chronic cardiopulmonary, liver, or renal disease; diabetes mellitus; alcoholism; malignancies; asplenia; immuno suppressive conditions or drugs; or use of antimicrobials in the prior 3 months (if so, select an alternative agent from a different class) A respiratory fluoroquinolone (oral moxifloxacin, 400 mg/day, or gemifloxacin, 320 mg/day, or levofloxacin, 750 mg daily for at least 5 days), or A  $\beta$ -lactam (e.g., ceftriaxone, 1-2 g IM each day for at least 5 days) plus a macrolide (e.g., azithromycin, 500 mg orally on day 1, followed by 250 mg orally each day on days 2-5)

3. In regions with a high rate (>25%) of infection with high-level (MIC  $\geq$ 16 mg/mL) macrolide-resistant *Streptococcus pneumoniae*, consider an alternative agent as noted under (2) for patients without comorbidities.

### **INPATIENTS, NON-ICU MANAGEMENT**

1. A respiratory fluoroquinolone (moxifloxacin, 400 mg/day, or gemifloxacin, 320 mg/day, or levofloxacin, 750 mg IV for 2 days followed by oral for at least 5 days total), or
2. A  $\beta$ -lactam (e.g., ceftriaxone 1-2 g IV daily for at least 1-2 days, followed by 1-2 g IM daily for at least 5 days total), plus A macrolide (azithromycin, 500 mg IV each day for at least 2 days, followed by 500 mg orally each day for a total of at least 5 days)

### **INPATIENTS—ICU MANAGEMENT**

1. A  $\beta$ -lactam (cefotaxime, 1-2 g IV every 6-8 hr, or ceftriaxone, 1-2 g IV each day, or ampicillin-sulbactam, 1.5-3 g IV every 6 hours, up to maximum of 4 g of sulbactam/day, for 7-14 days, plus Either azithromycin, 500 mg IV each day for at least 2 days, followed by 500 mg orally each day for a total of at least 5 days, or a respiratory fluoroquinolone (e.g. moxifloxacin, 400 mg/day, or gemifloxacin, 320 mg/day, or levofloxacin, 750 mg IV daily for 7-14 days)
2. For penicillin-allergic individuals, a respiratory fluoroquinolone (e.g., moxifloxacin, 400 mg/day, or gemifloxacin, 320 mg/day, or levofloxacin, 750 mg IV daily for 7-14 days) and aztreonam, 2 g IV every 6-8 hr for 7-14 days, are recommended.



## SPECIAL CONCERNS

If *Pseudomonas* species infection is a concern:

1. An antipneumococcal, antipseudomonal  $\beta$ -lactam (piperacillin-tazobactam, 3.375 g IV every 6 hr, or cefepime, 1-2 g every 12 hr, or imipenem, 500 mg every 6 hr or 1 g every 8 hr, or meropenem, 1 g IV every 8 hr), plus either ciprofloxacin, 400 mg IV every 8 hr, or levofloxacin, 500-750 mg IV every day, for 7-14 days, or
2. The above  $\beta$ -lactam plus an aminoglycoside (e.g., gentamicin, 7 mg/kg/day in three divided doses, with monitoring to maintain trough levels lower than 1  $\mu$ g/mL, or tobramycin, 7 mg/kg/day in three divided doses, with monitoring to maintain trough levels lower than 1  $\mu$ g/mL) and azithromycin (500 mg IV each day for at least 2 days, followed by 500 mg orally each day) for 7-14 days, or
3. The above  $\beta$ -lactam plus an aminoglycoside (as described above) and an antipneumococcal fluoroquinolone (ciprofloxacin, 400 mg IV every 8 hr, or levofloxacin, 500-750 mg IV every day) for 7-14 days For penicillin-allergic patients, use aztreonam, 2 g IV every 6-8 hr for 7-14 days, instead of the  $\beta$ -lactam.

If community-acquired methicillin-resistant *Staphylococcus aureus* is a consideration, add vancomycin, 15 mg/kg every 12 hr with monitoring to maintain trough at 15-20  $\mu$ g/mL, or linezolid, 600 mg every 12 hr, for 7-14 days<sup>44</sup>.

### **The duration of treatment for CAP**

Patients have usually been treated for 10–14 days, but recent studies with fluoroquinolones and telithromycin suggest that a 5-day course is sufficient for otherwise uncomplicated CAP. Most patients with CAP who are admitted to the hospital are treated with intravenous medications initially and then complete a 12-day oral course of therapy for a total of 14 days of combined intravenous and oral therapy<sup>72</sup>. A longer course is required for patients with bacteremia, metastatic infection, or infection with a particularly virulent pathogen, such as *P. aeruginosa* or CA-MRSA<sup>73</sup>. Longer-term therapy should also be considered if initial treatment was ineffective and in most cases of severe CAP. Patients may be discharged from the hospital once they are clinically stable and have no active medical problems requiring ongoing hospital care.

The site of residence after discharge (in a nursing home, at home with family, at home alone) is an important consideration, particularly for elderly patients.

### **General Considerations<sup>74</sup>**

Most experts feel that coverage should be divided against typical and atypical CAP pathogens<sup>75</sup>. In addition to appropriate antimicrobial therapy, certain general considerations apply in dealing with CAP. Adequate hydration, oxygen therapy for hypoxemia, and assisted ventilation when necessary are critical to the success of therapy. Patients with severe CAP who remain hypotensive despite fluid resuscitation may have adrenal insufficiency and may respond to glucocorticoid treatment.

## **Failure to Improve<sup>75</sup>**

Patients who are slow to respond to therapy should be reevaluated at about day 3 (sooner if their condition is worsening rather than simply not improving), and a number of possible scenarios should be considered.

1. Is this a noninfectious condition?
2. If this is an infection, is the correct pathogen being targeted?
3. Is this a superinfection with a new nosocomial pathogen?

A number of noninfectious conditions can mimic pneumonia.

They are

- Pulmonary edema,
- Pulmonary embolism,
- Lung carcinoma,
- Hypersensitivity pneumonitis, and
- Connective tissue disease involving the lungs.

If the patient has CAP and treatment is aimed at the correct pathogen, the lack of response may be explained in a number of ways.

The pathogen may be resistant to the drug selected <sup>76</sup>, or a sequestered focus (e.g., a lung abscess or empyema) may be blocking access of the antibiotic(s) to the pathogen.

Alternatively, the patient may be getting either the wrong drug or the correct drug at the wrong dose or frequency of administration<sup>77</sup>. It is also possible that CAP is the correct diagnosis but that a different pathogen (e.g., *M. tuberculosis* or a fungus) is the cause.

In addition, nosocomial superinfections—both pulmonary and extrapulmonary—are possible explanations for persistence<sup>31</sup>. In all cases of delayed response or deteriorating condition, the patient must be carefully reassessed and appropriate studies initiated. These studies may include such diverse procedures as CT and bronchoscopy

### **Complications<sup>5</sup>**

Complications in CAP depend on the infecting pathogen and patient health. For example, empyema can occur with *Streptococcus pneumoniae*, *Klebsiella pneumoniae*, and group A streptococcal CAP. (*K pneumoniae* infections occur in patients with chronic alcoholism.) Cavitation is not a feature of pneumococcal pneumonia, but it is a normal part of the disease process in *K pneumoniae* infections.

Myocardial infarction can be precipitated by fever due to community-acquired pneumonia (CAP).

Patients with CAP who have impaired splenic function may develop overwhelming pneumococcal sepsis, potentially leading to death within 12-24 hours, regardless of the antimicrobial regimen used.

As in other severe infections, common complications of severe CAP include

- 1) Respiratory failure,
- 2) Shock and
- 3) Multi-organ failure
- 4) Bleeding diatheses
- 5) Exacerbation of comorbid illnesses.
- 6) Metastatic infection
- 7) Lung abscess

8) Complicated pleural effusion.

9) Metastatic infection (e.g., brain abscess or endocarditis), although unusual, deserves immediate attention, with a detailed workup and proper treatment.

Lung abscess may occur in association with aspiration or with infection caused by a single CAP pathogen, such as CA-MRSA, *P. aeruginosa*, or (rarely) *S. pneumoniae*.

10) Myocardial infarction

In aspiration pneumonia, an infiltrate develops in a patient at increased risk of oropharyngeal aspiration. This occurs when a patient inhales material from the oropharynx that is colonized by upper airway flora.

The risk of aspiration is indirectly related to the level of consciousness of the patient (ie, decreasing Glasgow Coma score is related with increased risk of aspiration)<sup>77</sup>. Aspiration pneumonia is typically a mixed polymicrobial infection involving both aerobes and anaerobes and antibiotics that cover the known or suspected pathogens should be administered.

A significant pleural effusion should be tapped for both diagnostic and therapeutic purposes.

If the fluid has a

1. pH of <7,
2. a glucose level of <2.2 mmol/L, and
3. a lactate dehydrogenase concentration of >1000 U/L or
4. if bacteria are seen or cultured, then the fluid should be drained; a chest tube is usually required<sup>78</sup>.

## **Follow-Up**

Fever and leukocytosis usually resolve within 2 and 4 days, respectively, in otherwise healthy patients with CAP, but physical findings may persist longer. Chest radiographic abnormalities are slowest to resolve and may require 4–12 weeks to clear, with the speed of clearance depending on the patient's age and underlying lung disease. For a patient whose condition is improving and who (if hospitalized) has been discharged, a follow-up radiograph can be done 4–6 weeks later. If relapse or recurrence is documented, particularly in the same lung segment, the possibility of an underlying neoplasm must be considered.

## **Vaccines**

Pneumococcal vaccines prevent pneumococcal bacteremia but not necessarily pneumococcal pneumonia<sup>79</sup>. Two pneumococcal vaccines are approved in the United States. Prevnar 13, a pneumococcal 13-valent conjugate vaccine is approved for children aged 6 weeks to 5 years and adults aged 50 years or older. The 23-valent vaccine (Pneumovax 23) is approved for adults aged 50 years or older and persons aged 2 years or older who are at increased risk for pneumococcal disease.

On October 12, 2012, the Advisory Committee on Immunization Practices (ACIP) published updated recommendations for pneumococcal vaccination of high-risk adults. The committee now recommends routine use of Prevnar 13 in addition to the previously recommended Pneumovax 23 for adults aged 19 years and older with immuno-compromising conditions (eg, HIV, cancer, renal disease), functional or anatomic asplenia, cerebrospinal fluid leaks, or cochlear implants. Patients who have not previously received either vaccine should be given 1 dose of Prevnar 13 followed by 1

dose of Pneumovax 23 after at least 8 weeks. In patients who have previously received Pneumovax 23 vaccine, administer 1 dose of Prevnar 13 at least 1 year after the last Pneumovax 23 dose<sup>80</sup>.

On August 13, 2014, the ACIP recommended routine use of pneumococcal vaccine 13-valent (PCV13 [Prevnar 13]) in patients aged 65 years or older<sup>81</sup>. PCV13 should be administered in series with the 23-valent pneumococcal vaccine polyvalent (PPSV23 [Pneumovax23]), the vaccine currently recommended for adults aged 65 years or older. PCV13 was approved by the FDA in late 2011 for use among adults aged 50 years or older. In June 2014, the results of a randomized placebo-controlled trial evaluating efficacy of PCV13 for preventing community-acquired pneumonia among approximately 85,000 adults aged 65 years or older with no prior pneumococcal vaccination history (CAPiTA trial) became available and were presented to ACIP<sup>82</sup>.

## **MATERIALS AND METHODS**

### **SOURCE OF DATA**

Eighty patients who were diagnosed to be having community-acquired pneumonia admitted in R L Jalappa Hospital satisfying the aforementioned criteria were included in the study. The study was undertaken over the duration of one year.

Sample size has been calculated using the following formula where  $z_{crit}$  and  $z_{pwr}$  are cut-off points along the x axis of a standard normal probability distribution that demarcate probabilities matching the specified significance criterion and statistical power, respectively,  $p_1$  and  $p_2$  are pre-study estimates of the two proportions to be compared,

$D = (p_1 - p_2)$  (i.e., the minimum expected difference) and  $p = (p_1 + p_2)/2$ .

$$N = 2 \cdot [z_{crit} \sqrt{2\bar{p}(1 - \bar{p})} + z_{pwr} \sqrt{p_1(1 - p_1) + p_2(1 - p_2)}]^2 / D^2$$

A significance criterion of 0.05 and a power of 0.90 was chosen.

### **METHOD OF COLLECTION OF DATA**

Patients aged 18 years or more diagnosed with community acquired pneumonia on the grounds of the inclusion criteria were enrolled in the study. Those whose diagnosis changed during the course of treatment or who later fit into the exclusion criteria were excluded.



Patients were diagnosed as suffering from CAP if they have :-

- i. Fever or hypothermia, tachypnoea, cough with or without sputum, dyspnoea, chest discomfort, sweats or rigors (or both).
- ii. Bronchial breath sounds or inspiratory crackles on chest auscultation.
- iii. Parenchymal opacity on chest radiograph.
- iv. Symptoms occurred outside of the hospital or within 48 hours of hospital admission in a patient not residing in a long-term care facility.

At the time of initial evaluation, the selected patients underwent a complete clinical history and examination; chest radiograph (postero-anterior or antero-posterior views) at presentation; electrocardiogram; arterial blood gas analysis and serum electrolyte measurement; sputum for gram staining and culture; complete blood counts, blood urea nitrogen and serum creatinine; fasting blood glucose.

For performing ABG (arterial blood gas analysis) analysis best care practices were followed. Precautions set by the institution to be used while handling body fluids were followed strictly. Modified Allen's test was performed in the limb selected for the procedure. The patient's radial pulse was palpated with the index and middle finger pads of the non-dominant hand. After visualizing the direction of the artery, and the desired puncture site was cleaned. The needle was inserted just under the skin at a 45° angle, aiming in the direction of the artery, while palpating the radial pulse proximal to the puncture site with the non-dominant hand. After 2-3 mL of arterial blood has been obtained, the needle was removed.

As required patients were connected to mechanical ventilatory support and tidal volume (6ml/kg body weight) and PEEP was set according to patient's need to maintain oxygen Saturation (88-95%), plateau pressures (<30cmH<sub>2</sub>O) and respiratory rate <35/min according to standard treatment protocol.

A questionnaire with demographic information, clinical signs and symptoms, laboratory and radiographic findings was completed for each patient. Each patient was assessed with both the scoring systems and total score for each patient for each scoring system was calculated. The patients' clinical outcome was also recorded within two weeks after admission.



**Figure 7: Iontropic and Ventilatory Support being given to a patient of CAP**



**A**



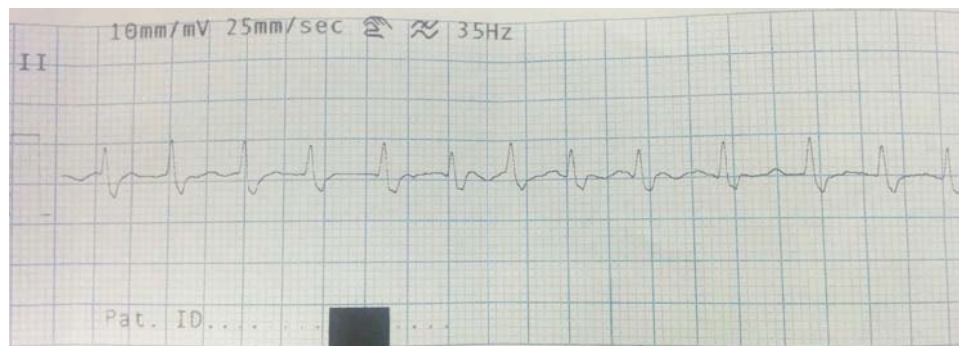
**B**

**Figure 8 : A- Arterial Blood Gas Analyzer(ABL 80 Flex)**

**B - Depiction of Femoral and Radial Arterial Blood Sampling**



**A**



**B**

**Figure 9: A - Radiographic Depiction of Bilateral Homogenous Consolidation**

**suggestive of CAP**

**B – ECG showing Sinus Tachycardia**

## **INCLUSION CRITERIA**

1. Age more than 18yrs
2. Patients with clinical diagnosis of Pneumonia and chest radiograph consistent with diagnosis of Pneumonia.

## **EXCLUSION CRITERIA**

1. Chronically immunosuppressed patients (patients on steroids ,neutropaenic patients, immunosuppressive agents)
2. Patients hospitalized within previous 14 days
3. Patients with alternate diagnosis during follow up
4. Patients diagnosed with chronic obstructive pulmonary disease.

## **Investigations conducted on patients**

1. Chest radiograph (postero-anterior or antero-posterior view) at presentation;
2. Electrocardiogram;
3. Arterial blood gas analysis and serum electrolyte measurement;
4. Complete blood counts,
5. Blood urea and serum creatinine;
6. Fasting blood glucose.

## **STUDY DESIGN**

It is a Hospital-based prospective observational study.

## **STATISTICAL METHODS:**

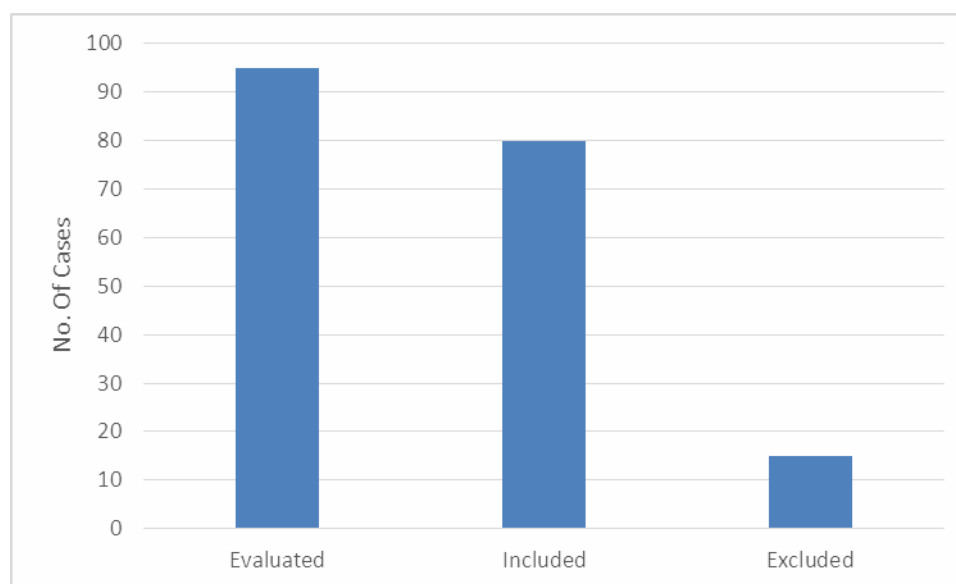
Data was entered into Microsoft excel data sheet and was analysed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Chi-square was used as test of significance. Continuous data was represented as mean and SD. Independent t test was used as test of significance to identify the mean difference between two groups. p value  $<0.05$  was considered as statistically significant. ROC curve was plotted to find the area under curve and sensitivity and specificity of PSI and CURB – 65 Score with respect to ICU Stay, Requirement of Ventilator use and Mortality.

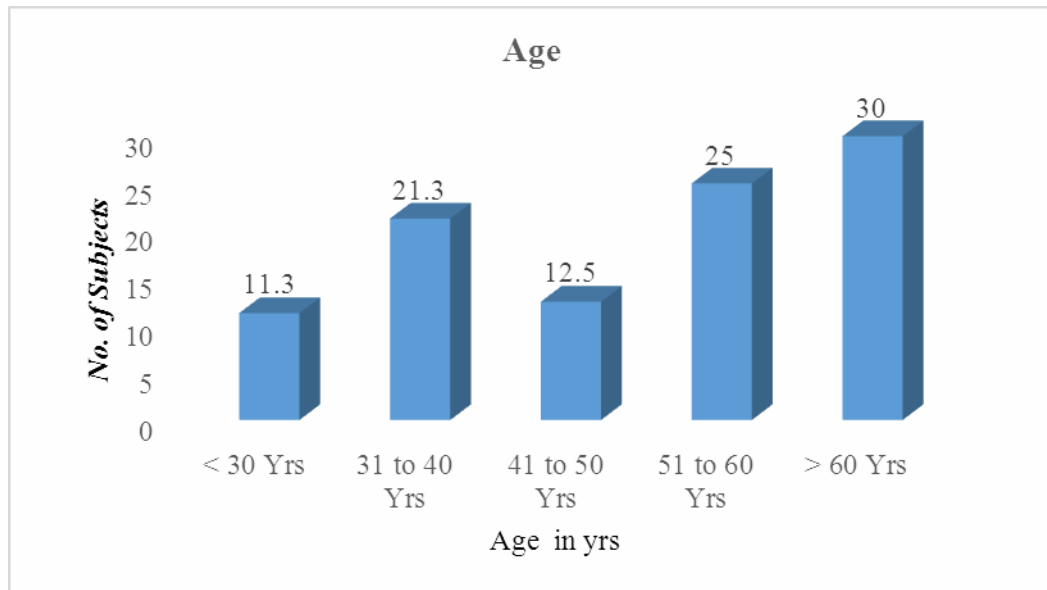
## RESULTS

In the study a total of 95 cases were evaluated. 80 subjects with Community Acquired Pneumonia (CAP) was included in the study who satisfied the inclusion criteria. 15 cases were excluded in accordance with the exclusion criteria.

**Table 9: Age Distribution of subjects**

Age Distribution	Frequency	Percent
< 30 Yrs	9	11.3
31 to 40 Yrs	17	21.3
41 to 50 Yrs	10	12.5
51 to 60 Yrs	20	25.0
> 60 Yrs	24	30.0
<b>Total</b>	<b>80</b>	<b>100.0</b>





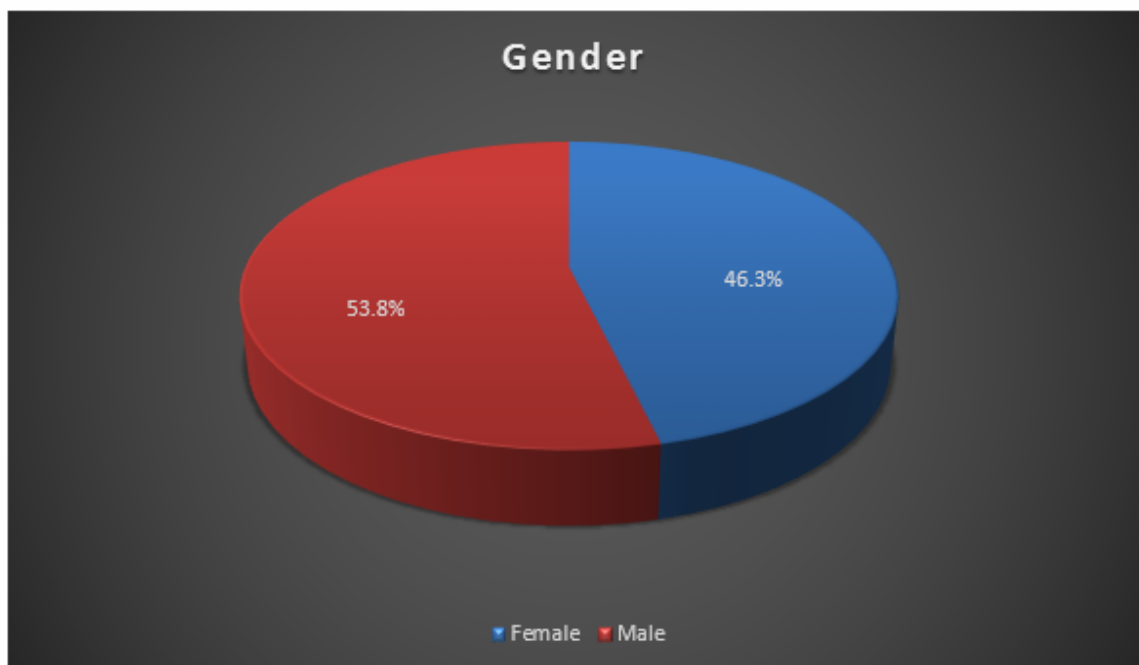
**Graph 1: Bar diagram showing Age distribution of subjects**

In the study Majority of subjects (70%) were less than 60 yrs of age with 30% in the age group > 60 years. 25% were in 51 to 60 years. Least no of subjects were in the age group < 30 years.



**Table 10: Gender distribution of subjects**

Gender	Frequency	Percent
Female	37	46.3
Male	43	53.8
Total	80	100.0

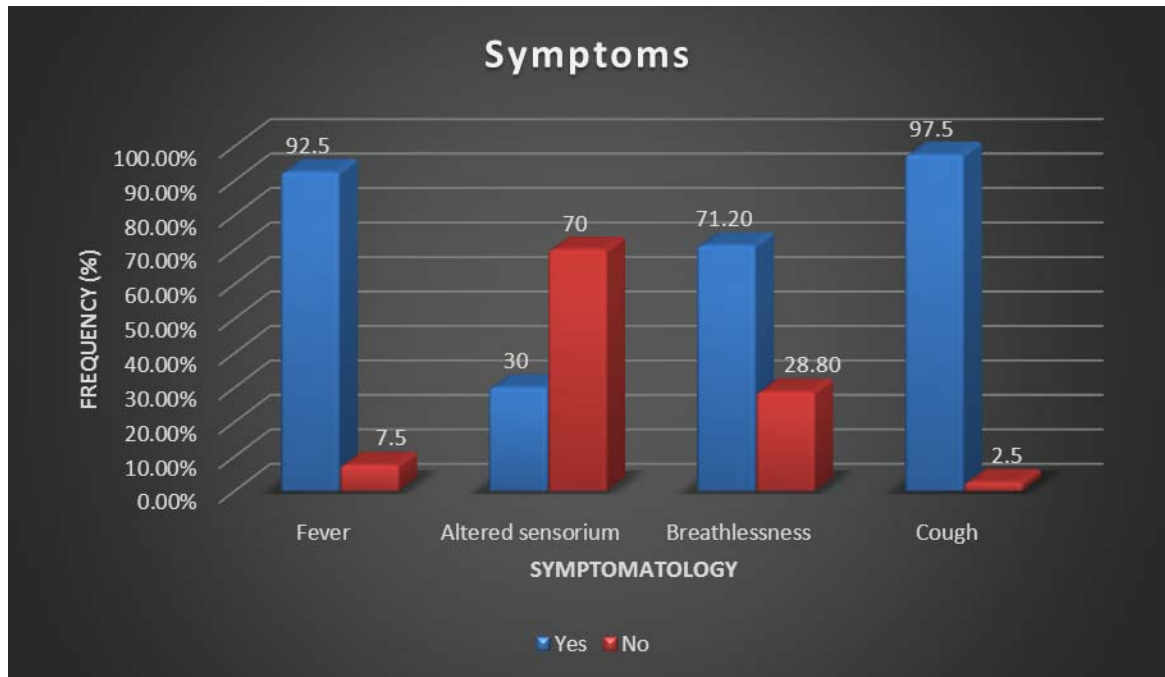


**Graph 2: Pie diagram showing Gender distribution of subjects  
(in Percentages)**

Majority (53.8%) of subjects were males and 46.3% were females. This is similar to the demographic distribution of people with respect to gender in the study population (adults residing in Kolar district).

**Table 11: Symptoms in Subjects**

Symptomatology	Yes	Percent	No	Percent
<b>Fever</b>	74	92.5%	6	7.5%
<b>Altered sensorium</b>	24	30.0%	56	70.0%
<b>Breathlessness</b>	57	71.2%	23	28.7%
<b>Cough</b>	78	97.5%	2	2.5%

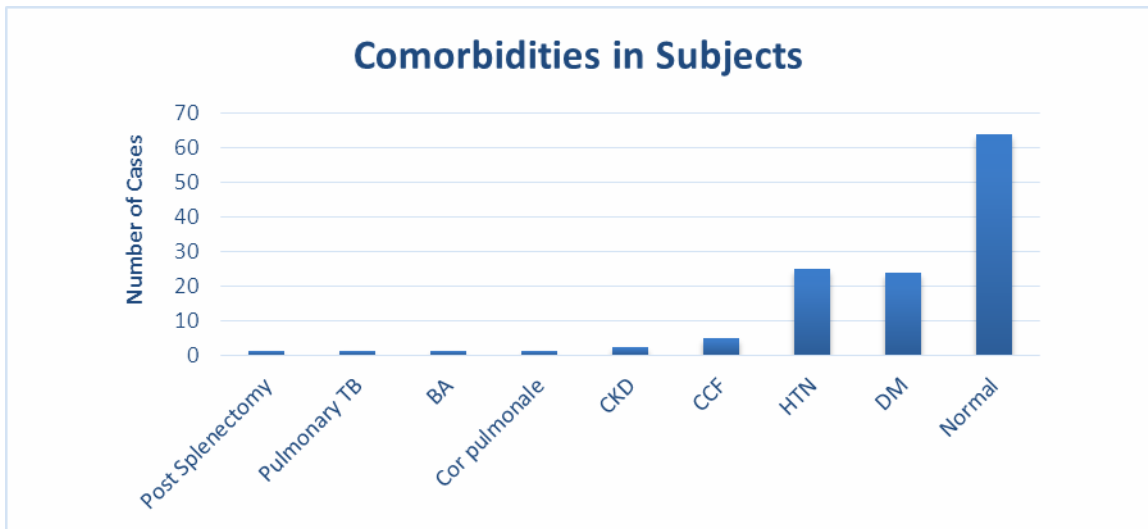


**Graph 3: Bar diagram showing Symptoms in Subjects**

In the study 92.5% had fever, 30% had altered sensorium, 71.2% had breathlessness and 97.5% had cough, among which 91% had expectoration and 15% had haemoptysis. Fever and cough with expectoration were thus the most common symptoms of CAP in our study.

**Table 12: Comorbidities in Subjects**

Comorbidities	Frequency	Percent (%)
Devoid of any comorbidities	51	63.7
DM	19	23.7
HTN	20	25
CCF	4	5
CKD	2	2.5
Cor pulmonale	1	1.3
BA	1	1.3
Pulmonary TB	1	1.3
Post Splenectomy	1	1.3



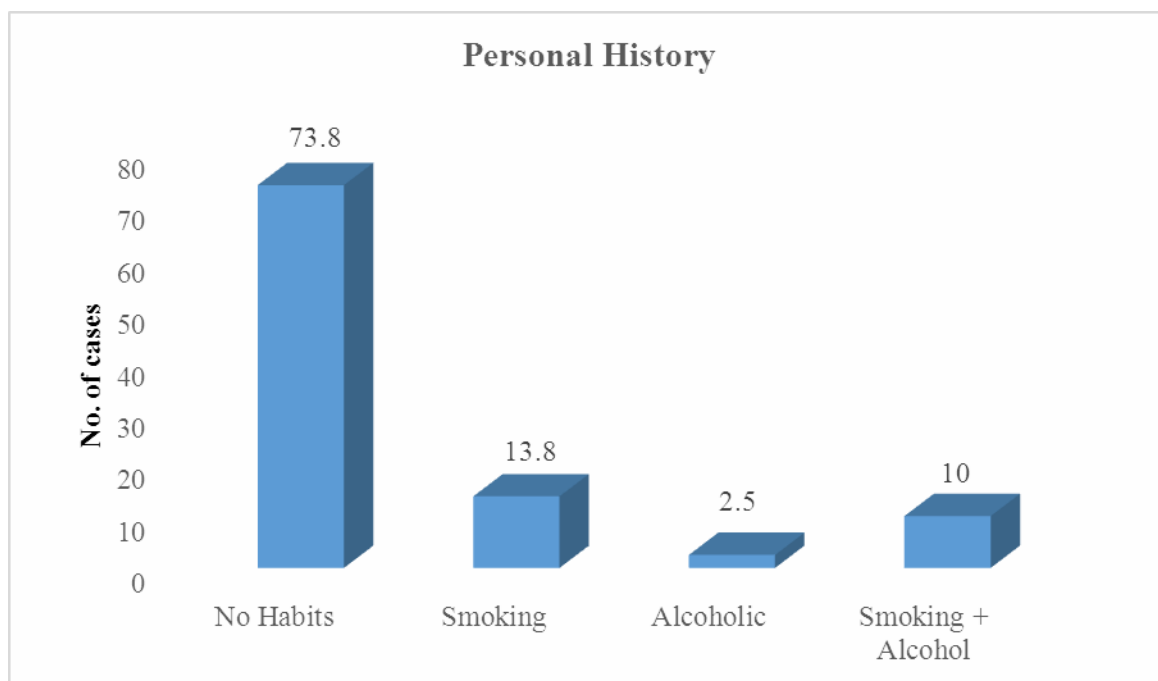
**Graph 4: Comorbidities in Subjects**

In our study 23.7% had diabetes mellitus, 25% had HTN, 5% had CCF, 2.5% had CKD and 1.3% had Cor pulmonale, Bronchial asthma (BA), Pulmonary TB and splenectomy respectively, thus diabetes mellitus and hypertension were the most common associated comorbidities.

**Table 13: Personal History of subjects**

Personal History	Frequency	Percent
No Habits	59	73.8
Smoking	11	13.8
Alcoholic	2	2.5
Smoking & Alcohol	8	10.0
Total	80	100.0

**Graph 5: Bar diagram showing Personal History of subjects**

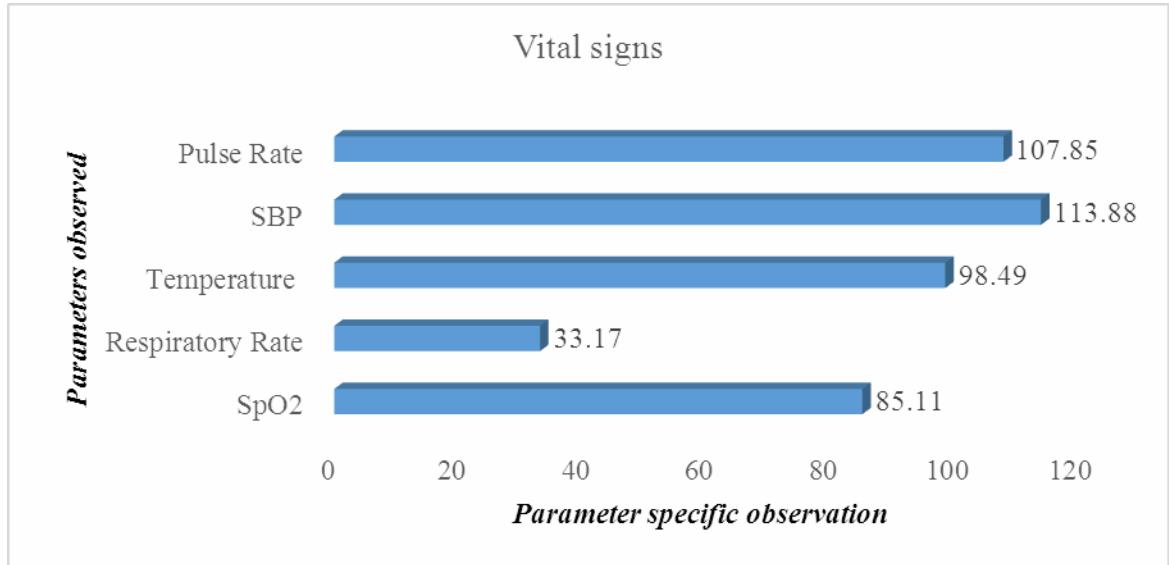


Smoking history was present in 13.8% of subjects and 2.5% were alcoholic and 10% were both alcoholic and Smokers.

**Table 14: Mean and SD of Various Parameters**

<b>Vital Parameters</b>	<b>Mean</b>	<b>SD</b>
<b>Pulse Rate</b>	107.85	21.35
<b>SBP</b>	113.88	30.43
<b>Temperature</b>	98.49	2.29
<b>Respiratory Rate</b>	33.17	11.00
<b>SpO2</b>	85.11	17.18
<b>pH</b>	7.28	0.18
<b>PaO2</b>	83.71	29.90
<b>PaCo2</b>	46.76	24.76
<b>B.Urea</b>	55.43	39.94
<b>BUN</b>	25.90	18.66
<b>S.Creatinine</b>	2.12	2.68
<b>Serum Sodium</b>	132.57	6.99
<b>Serum Potassium</b>	4.40	0.97
<b>Blood Glucose</b>	162.36	101.91
<b>Serum Albumin</b>	3.21	0.75

The above tables mean and standard deviation of various quantitative parameters in the study. Variations in the Systolic BP, Respiratory Rate, pH and PaO2 were directly related to the outcome of the patient with poor outcome associated with both the extremes of distribution.

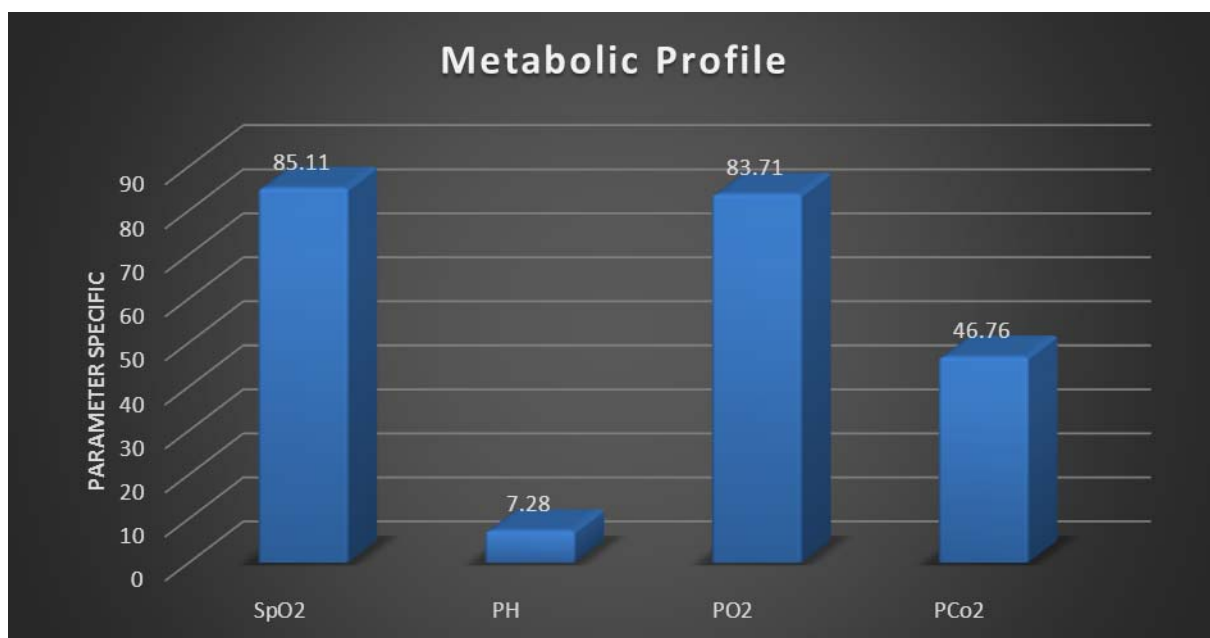


**Graph 6: Bar diagram showing Vital signs**

The average Pulse Rate obtained in the cases was 107.85 per minute i.e. Tachycardia was observed in the majority of the patients presenting with CAP.

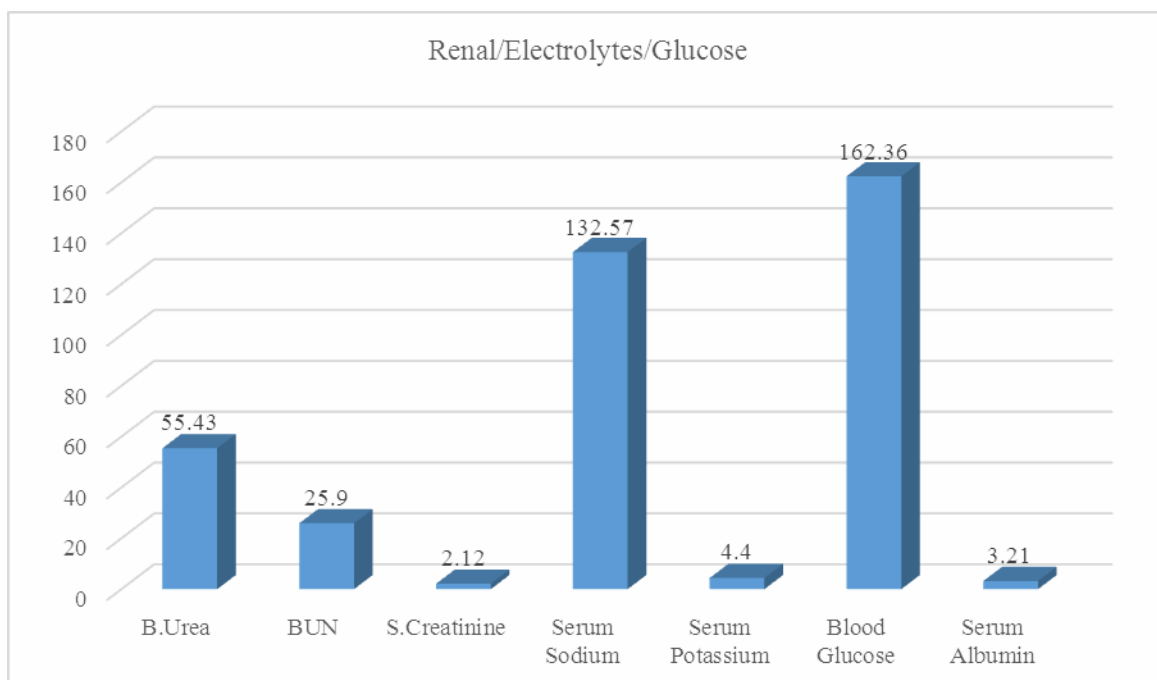
An increased Respiratory Rate with an average of 33.17 cycles per minute was observed i.e. Tachypnea was present in majority of cases of CAP. The mean arterial oxygen saturation of the patients at the time of presentation was 85.11%, which is 11% less than the lower limit of the normal range (96 -100 %).

Mean axillary temperature is within the normal limits, thus making it a poor marker for the severity of CAP, although an association was noted between low systolic blood pressure and low axillary temperature.



**Graph 7: Bar diagram showing Metabolic Profile of subjects**

Similarly mean pH observed was 7.28 which was less than the average (7.35 – 7.45). Partial pressure of oxygen ( $\text{PaO}_2$  or  $\text{pO}_2$ ) was noted within its standardised range of 75 - 100 mmHg, i.e. an average of 83.71 mmHg. The Partial pressure of  $\text{CO}_2$  ( $\text{PaCO}_2$  or  $\text{pCO}_2$ ) was observed with an average value of 46.76 which was more than the standard range 35-45 mmHg.



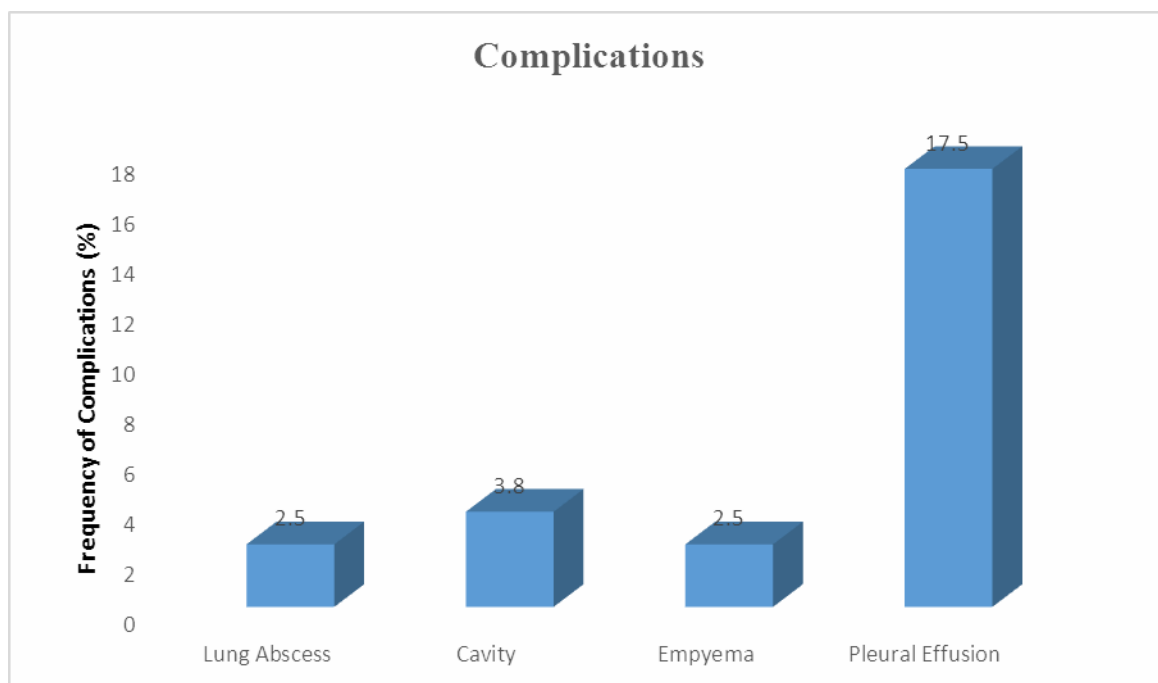
**Graph 8: Bar diagram showing Renal/ Electrolyte and Glycemic Profile of subjects**

The Renal parameters of the patients were deranged with average Blood Urea, Blood Urea Nitrogen (BUN) and Serum Creatinine values elevated above the normal levels. Serum electrolytes (Sodium and Potassium) were noted in the normal range and Random Blood glucose was noted in the higher range probably attributable to the high number of patients having diabetes mellitus as the comorbidity.



**Table 15: Complications in Community Acquired Pneumonia cases**

Complications	Yes	%	No	%
Lung Abscess	2	2.5	78	97.5
Cavity	3	3.8	77	96.3
Empyema	2	2.5	78	97.5
Pleural Effusion	14	17.5	66	82.5

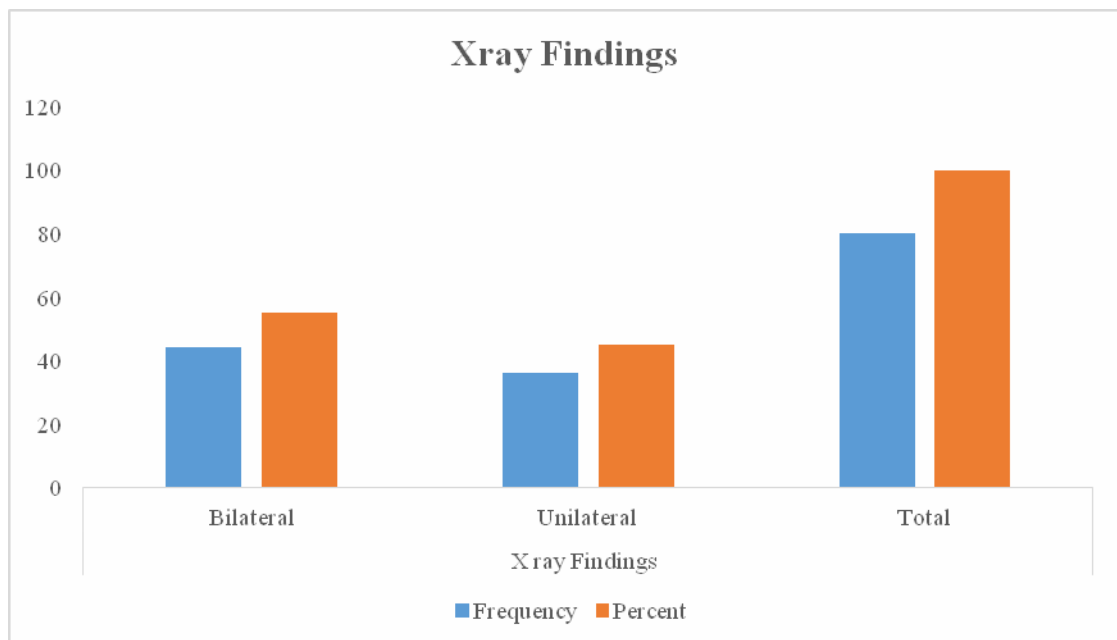


**Graph 9: Bar diagram showing Complications in Community Acquired Pneumonia cases (in Percentages)**

In the study 2.5% had lung abscess and Empyema respectively, 3.8% had cavity and 17.5% had pleural effusion also called as synpneumonic pleural effusion, thus making pleural effusion as the most common complication associated with pneumonia in our study.

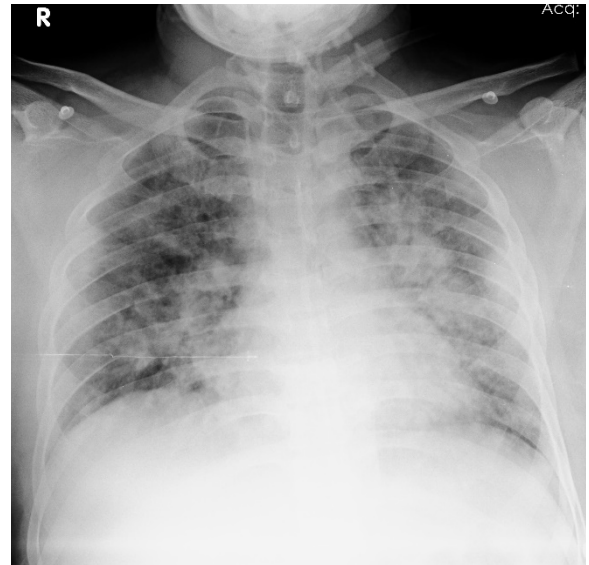
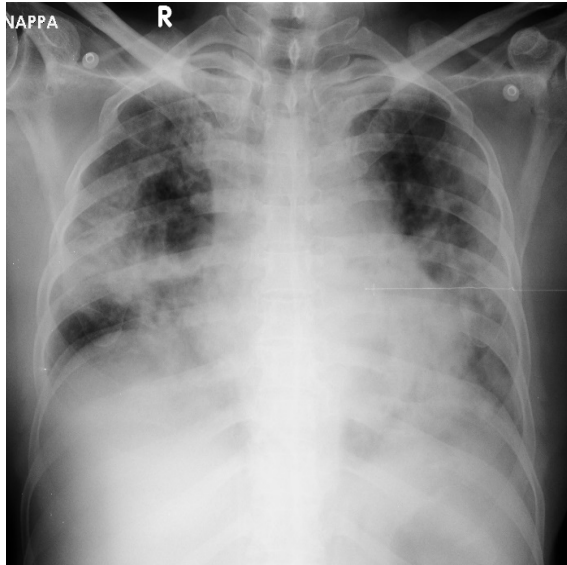
**Table 16: X- ray Findings of Lobe Involved in CAP subjects**

X ray Findings	Frequency	Percent (%)
Unilobular	36	45
Multilobular	44	55
Total	80	100



**Graph 10: X- ray Findings of Lobe Involved in CAP subjects**

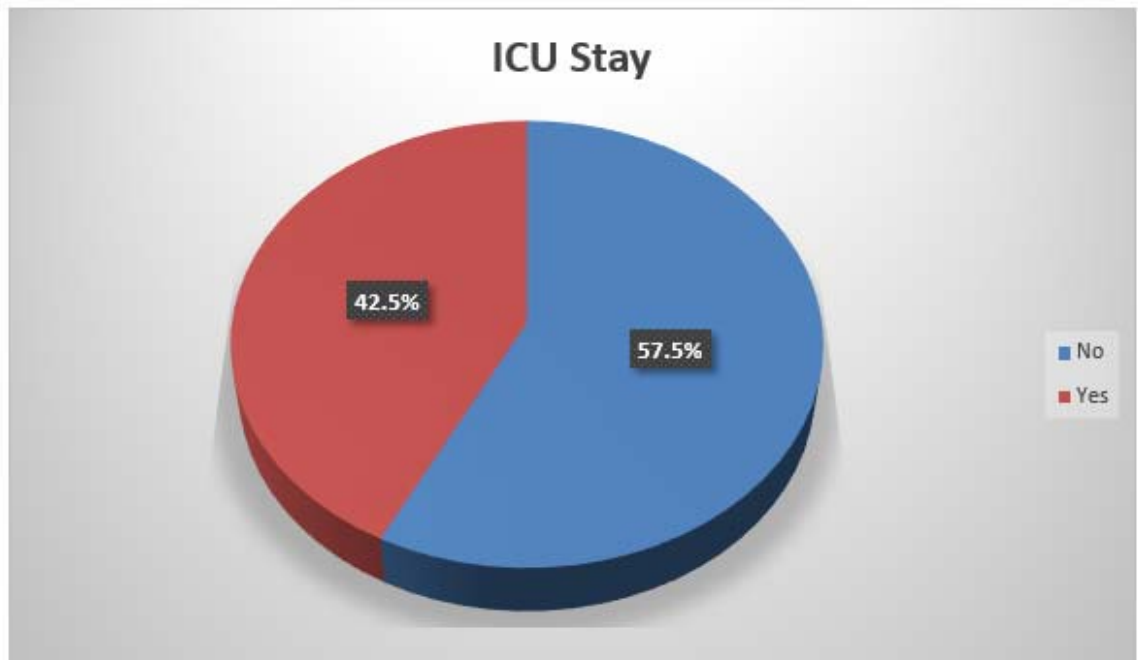
Majority of subjects suffering from CAP i.e. 55 % had multilobular involvement of the lung fields and 45% had unilobular involvement. Multilobular involvement is known to one of the High risk markers in CAP.



**Figure 10: Few Radiographs Depicting Various Patterns Of CAP**

**Table 17: Distribution of subjects according to ICU stay**

ICU Stay	Frequency	Percent (%)
Yes	34	42.5
No	46	57.5
Total	80	100

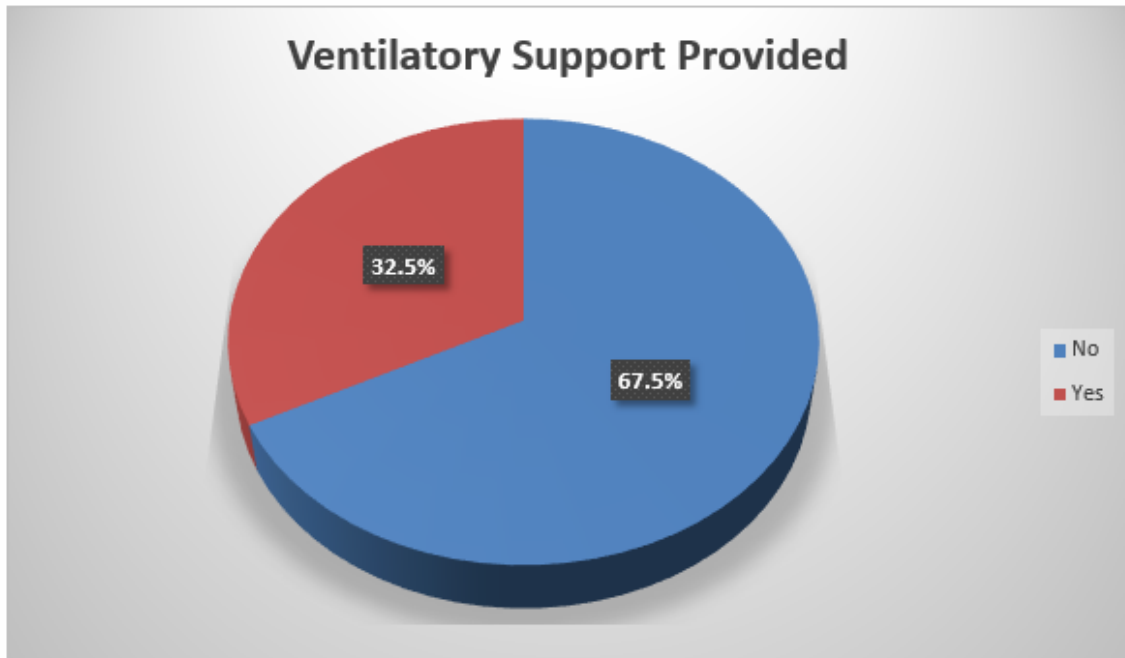


**Graph 11: Pie diagram showing ICU admission in CAP subjects (in Percentages)**

In the study 34 subjects (42.5%) were admitted in ICU. Although in our majority of cases did not require admission to Intensive Care Units.

**Table 18: Distribution of subjects according to Ventilator support**

Ventilator support	Frequency	Percent
No	54	67.5
Yes	26	32.5
Total	80	100.0

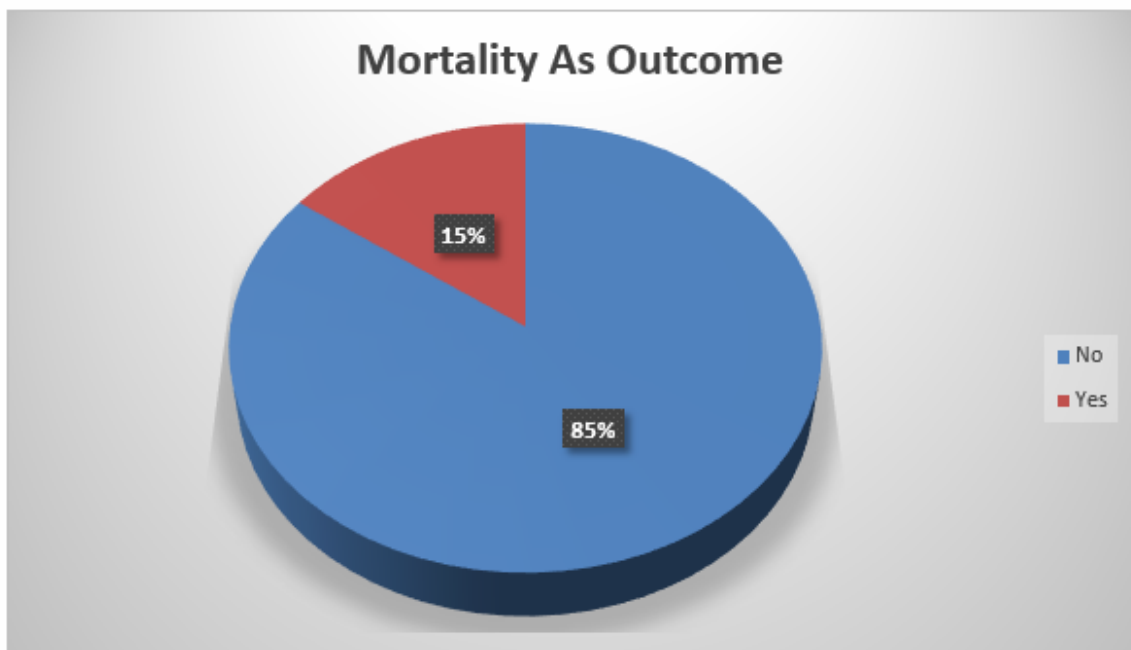


**Graph 12: Pie diagram showing Distribution of subjects according to Ventilatory support provided (in Percentages)**

In the study 32.5% of subjects were put on ventilator support. Although the majority of the cases though (67.5%) did not require ventilatory support.

**Table 19: Distribution of subjects according to Mortality**

<b>Mortality as Outcome</b>	<b>Frequency</b>	<b>Percent</b>
<b>No</b>	68	85.0
<b>Yes</b>	12	15.0
<b>Total</b>	80	100.0

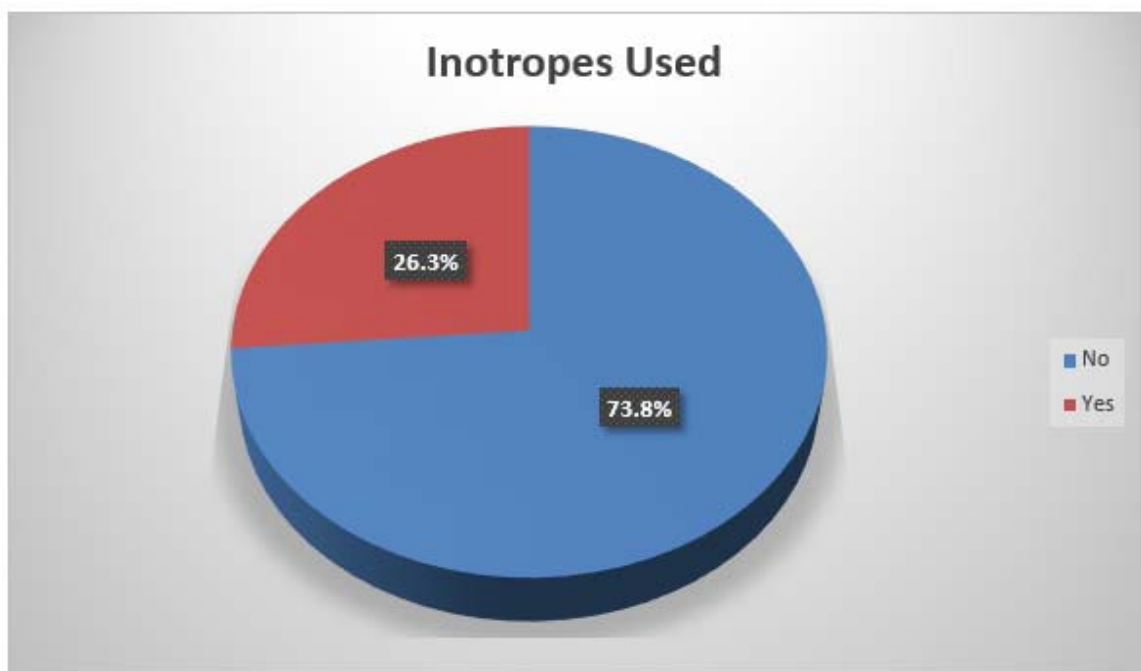


**Graph 13: Pie diagram showing Distribution of subjects according to Mortality  
(In Percentages)**

In the study 12 subjects (15%) of CAP had mortality. Good outcome as recovery (85%) was observed in the majority of the cases.

**Table 20: Distribution of subjects according to Inotropes used**

<b>Inotropes Used</b>	<b>Frequency</b>	<b>Percent</b>
<b>No</b>	59	73.8
<b>Yes</b>	21	26.3
<b>Total</b>	80	100.0

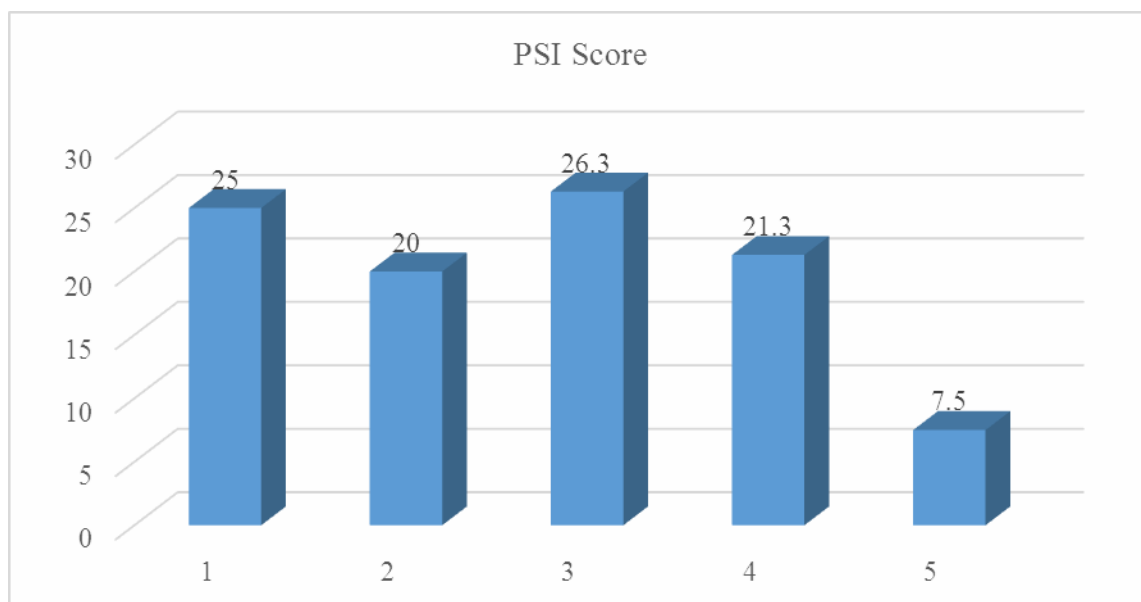


**Graph 14: Pie diagram showing Distribution of subjects according to Inotropes used**

21 subjects (26.3%) were put on Inotropes. Majority of the cases though (73.8%) although did not require ionotropic support.

**Table 21: Distribution of PSI Score in CAP subjects**

PSI Score	Frequency	Percent
<b>1</b>	20	25.0
<b>2</b>	16	20.0
<b>3</b>	21	26.3
<b>4</b>	17	21.3
<b>5</b>	6	7.5
<b>Total</b>	80	100.0



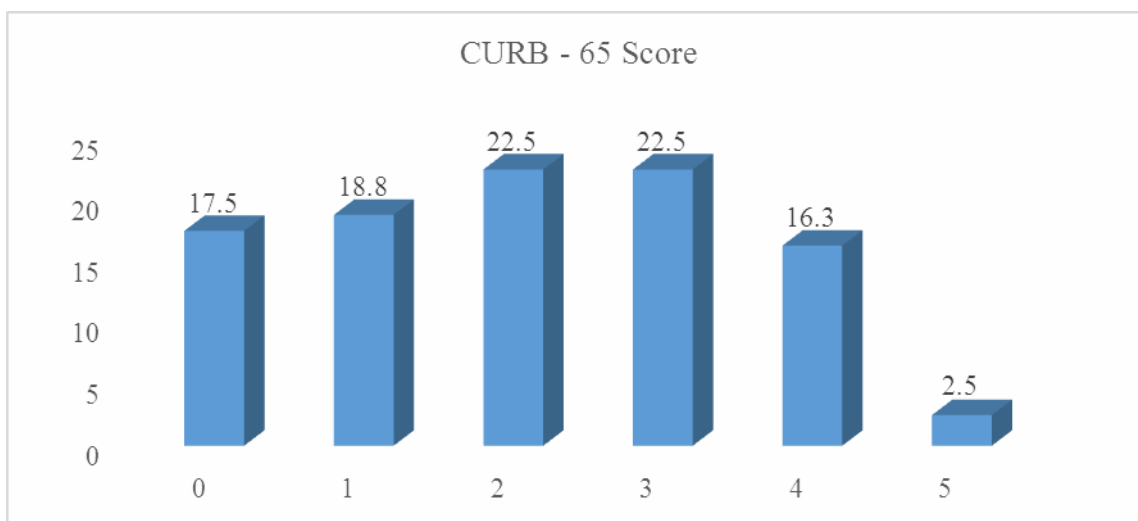
**Graph 15: Bar diagram showing Distribution of PSI Score in CAP subjects**

In our study 25% of subjects had 1 score, 20% had score 2, 26.3% had score 3, 21.3% had score 4 and 7.5% had score 5 PSI. Age was the most significant parameter in the PSI scoring system.



**Table 22: Distribution of CURB – 65 Score in CAP subjects**

<b>CURB – 65 Score</b>	<b>Frequency</b>	<b>Percent</b>
<b>0</b>	14	17.5
<b>1</b>	15	18.8
<b>2</b>	18	22.5
<b>3</b>	18	22.5
<b>4</b>	13	16.3
<b>5</b>	2	2.5
<b>Total</b>	80	100.0



**Graph 16: Bar diagram showing Distribution of CURB – 65 Score in CAP subjects**

In the study 17.5% had subjects had 0 CURB – 65 score, 18.8% had score 1, 22.5% had score 2 and Score 3 respectively, 16.3% had score 4 and 2.5% had score 5.

**Table 23: Association between CURB – 65 score with various parameters**

		CURB – 65 Score												P value
		0		1		2		3		4		5		
		Count	%	Count	%	Count	%	Count	%	Count	%	Count	%	
Age	Mean ± SD	43.29 ±10.06		45.73±15.43		54.89± 13.26		49.22±17.57		63.54±19.20		70.00±7.07		0.005*
Sex	Female	6	42.9%	6	40.0%	11	61.1%	9	50.0%	5	38.5%	0	0.0%	0.544
	Male	8	57.1%	9	60.0%	7	38.9%	9	50.0%	8	61.5%	2	100.0%	
Ventilator Support		1	7.1%	3	20.0%	3	16.7%	8	44.4%	9	69.2%	2	100.0%	0.001*
Inotropes Support		0	0.0%	0	0.0%	2	11.1%	10	55.6%	7	53.8%	2	100.0%	0.001*
ICU stay		1	7.1%	3	20.0%	9	50.0%	10	55.6%	9	69.2%	2	100.0%	0.002*
Death		0	0.0%	0	0.0%	0	0.0%	2	11.1%	8	61.5%	2	100.0%	0.001*

In the study when CURB – 65 score was compared with various parameters it was observed that there was significant association between Age, on ventilator support, on inotropes, ICU stay and Mortality. i.e. Higher CURB – 65 scores were seen in > 60 years subjects, patients who were put on ventilator, patients on inotropes, patients in ICU and patients who had Mortality (The p values obtained for all the parameters was within the set limits of statistically significance ie <0.05).

**Table 24: Association between PSI score with various parameters**

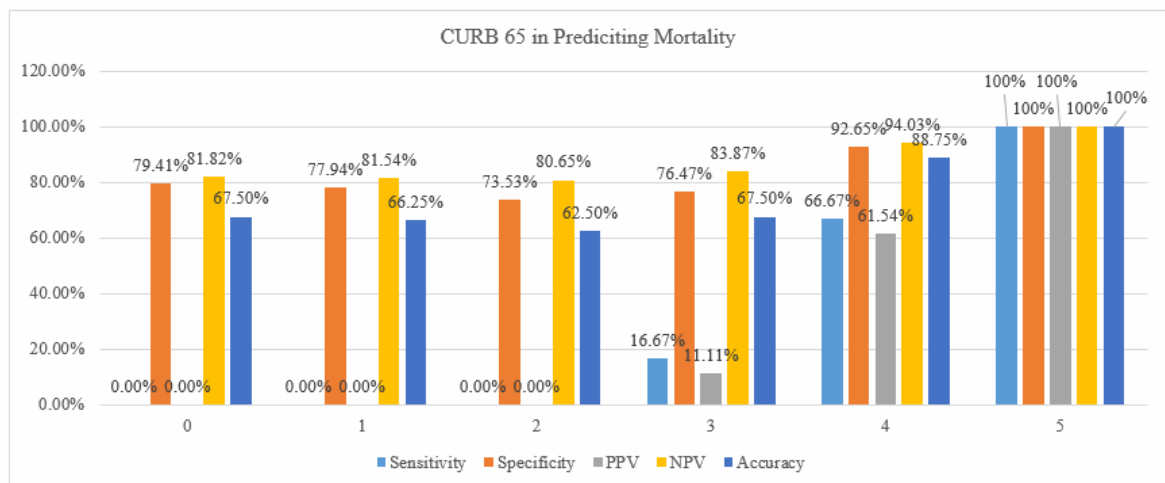
		CURB – 65 Score												P value
		0		1		2		3		4		5		
		Count	%	Count	%	Count	%	Count	%	Count	%	Count	%	
Age	Mean ± SD	43.29 ±10.06		45.73±15.43		54.89± 13.26		49.22±17.57		63.54±19.20		70.00±7.07		0.005*
Sex	Female	6	42.9%	6	40.0%	11	61.1%	9	50.0%	5	38.5%	0	0.0%	0.544
	Male	8	57.1%	9	60.0%	7	38.9%	9	50.0%	8	61.5%	2	100.0%	
Ventilator Support		1	7.1%	3	20.0%	3	16.7%	8	44.4%	9	69.2%	2	100.0%	0.001*
Inotropes Support		0	0.0%	0	0.0%	2	11.1%	10	55.6%	7	53.8%	2	100.0%	0.001*
ICU stay		1	7.1%	3	20.0%	9	50.0%	10	55.6%	9	69.2%	2	100.0%	0.002*
Death		0	0.0%	0	0.0%	0	0.0%	2	11.1%	8	61.5%	2	100.0%	0.001*

In the study when PSI score was compared with various parameters, it was observed that there was significant association between Age, on ventilator support, on inotropes, ICU stay and Mortality. i.e. Higher PSI scores were seen in > 60 years subjects, patients who were put on ventilator, patients on inotropes, patients in ICU and patients who had Mortality (The p values obtained for all the parameters was within the set limits of statistical significance ie <0.05).

	Score	Sensitivity	Specificity	PPV	NPV	Accuracy
<b>CURB – 65</b>	<b>0</b>	0.0%	79.41%	0.0%	81.82%	67.5%
	<b>1</b>	0.0%	77.94%	0.0%	81.54%	66.25%
	<b>2</b>	0.0%	73.53%	0.0%	80.65%	62.5%
	<b>3</b>	16.67%	76.47%	11.11%	83.87%	67.5%
	<b>4</b>	66.67%	92.65%	61.54%	94.03%	88.75%
	<b>5</b>	100%	100%	100%	100%	100%

**Table 25: Sensitivity, Specificity, PPV, NPV and accuracy of CURB 65 for predicting Mortality**

The above table shows sensitivity and specificity of CURB-65 in predicting mortality. Higher sensitivity and specificity was observed in CURB 65-score 4 and 5. I.e. higher CURB -65 scores had better diagnostic accuracy in diagnosing Mortality. This Graph shows that the Sensitivity, Specificity, PPV, NPV and accuracy of CURB 65 for predicting Mortality increases with increase in the class of the scoring system, i.e. higher the class, higher is the Sensitivity, Specificity, PPV, NPV and accuracy of CURB 65 Scoring system.

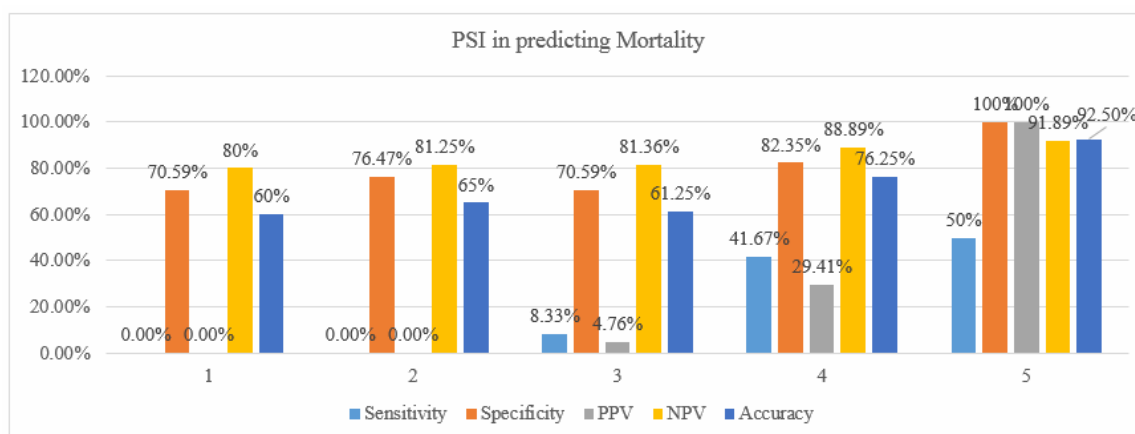


**Chart 17: Bar diagram showing Sensitivity, Specificity, PPV, NPV and accuracy of CURB 65 for predicting Mortality**

	Score	Sensitivity	Specificity	PPV	NPV	Accuracy
<b>PSI</b>	1	0.0%	70.59%	0.0%	80%	60%
	2	0.0%	76.47%	0.0%	81.25%	65%
	3	8.333%	70.59%	4.762%	81.36%	61.25%
	4	41.67%	82.35%	29.41%	88.89%	76.25%
	5	50%	100%	100%	91.89%	92.5%

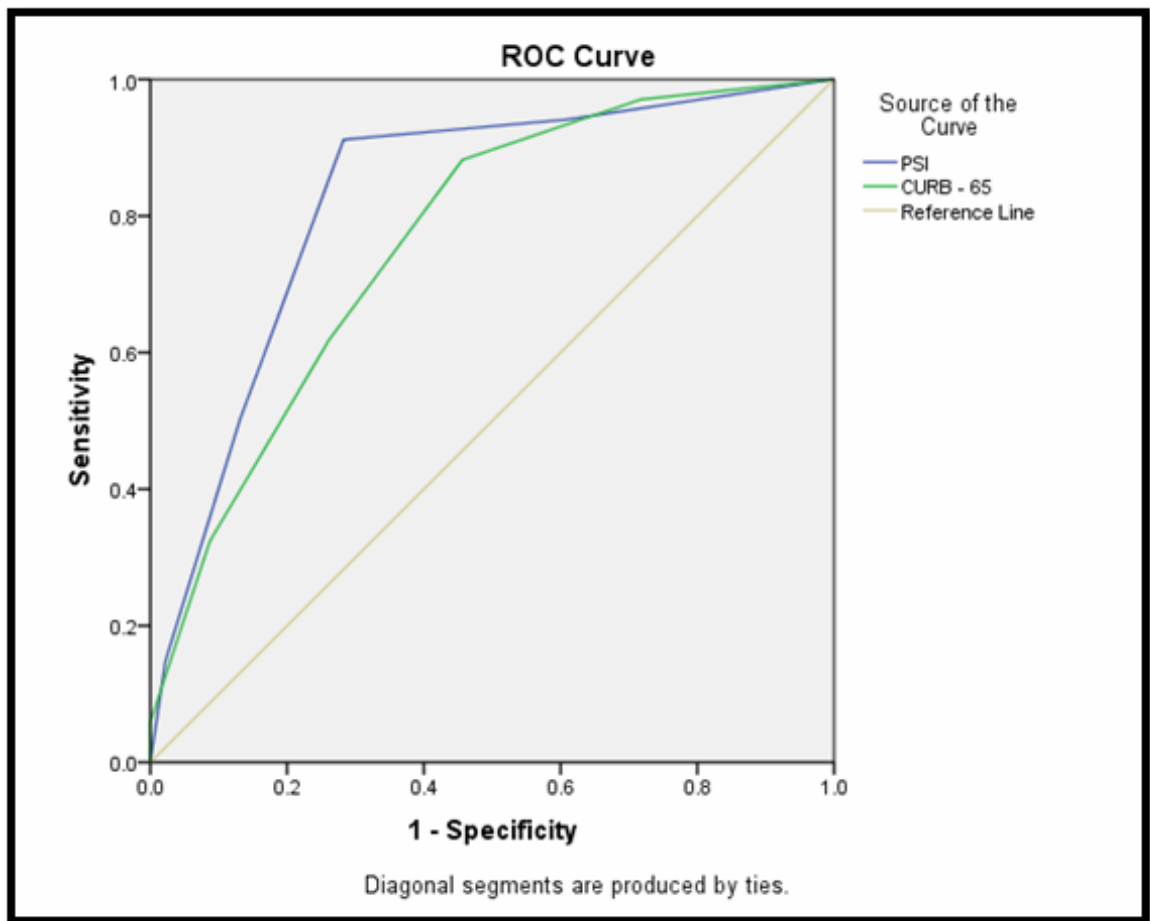
**Table 26: Sensitivity, Specificity, PPV, NPV and accuracy of PSI for predicting Mortality**

The above table shows sensitivity and specificity of PSI score in predicting mortality. Higher sensitivity and specificity was observed in PSI score 4 and 5. I.e. higher PSI scores had better diagnostic accuracy in diagnosing Mortality. This Graph shows that the Sensitivity, Specificity, PPV, NPV and accuracy of PSI Scoring system for predicting Mortality increases with increase in the class of the scoring system, i.e. higher the class, higher is the Sensitivity, Specificity, PPV, NPV and accuracy of PSI Scoring system.



**Chart 18: Bar diagram showing Sensitivity, Specificity, PPV, NPV and accuracy of PSI for predicting Mortality**

## Need for Admission to ICU



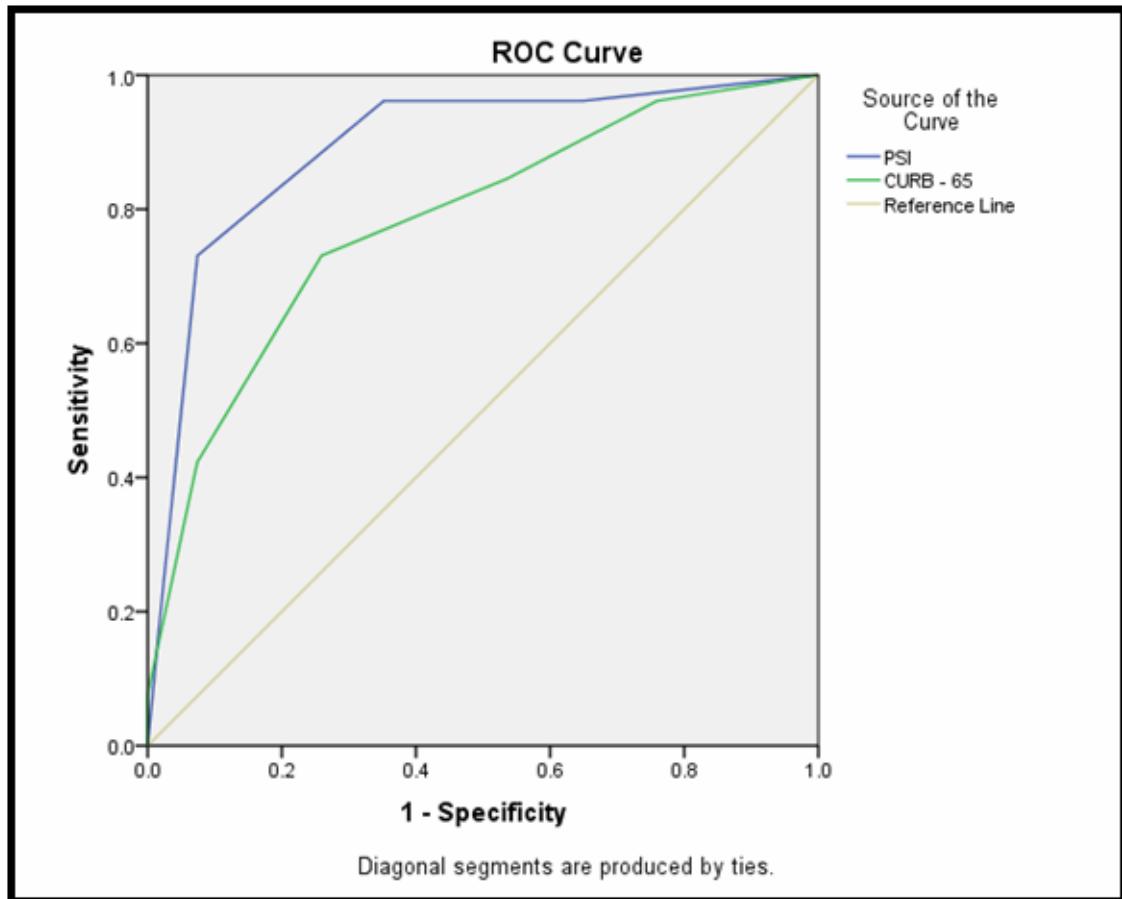
Graph 19: ROC Curve - Need for Admission to ICU

Test Result Variable(s)	Optimal Cut Off Score	Sensitivity	Specificity	Area Under the Curve	P value	95% CI	
						Lower Bound	Upper Bound
<b>PSI</b>	>2	0.912	0.717	0.826	<0.0001*	0.732	0.920
<b>CURB - 65</b>	>1	0.882	0.543	0.765	<0.0001*	0.662	0.869

**Table 27: Sensitivity, Specificity and Area under Curve (AUC) of PSI and CURB65 for Need for Admission to ICU**

In our study 34 subjects were admitted to ICU. Area under the curve for ICU admitted patients was highest for PSI score than CURB-65. I.e. PSI score has higher prediction for ICU admission in CAP patients. The p value obtained is within the parameters of significance and hence the association with both the CURB-65 score and PSI score with the need for admission to ICU is significant.

## Need for ventilation



**Graph 20: ROC Curve - Need for Need For Ventilatory Support**

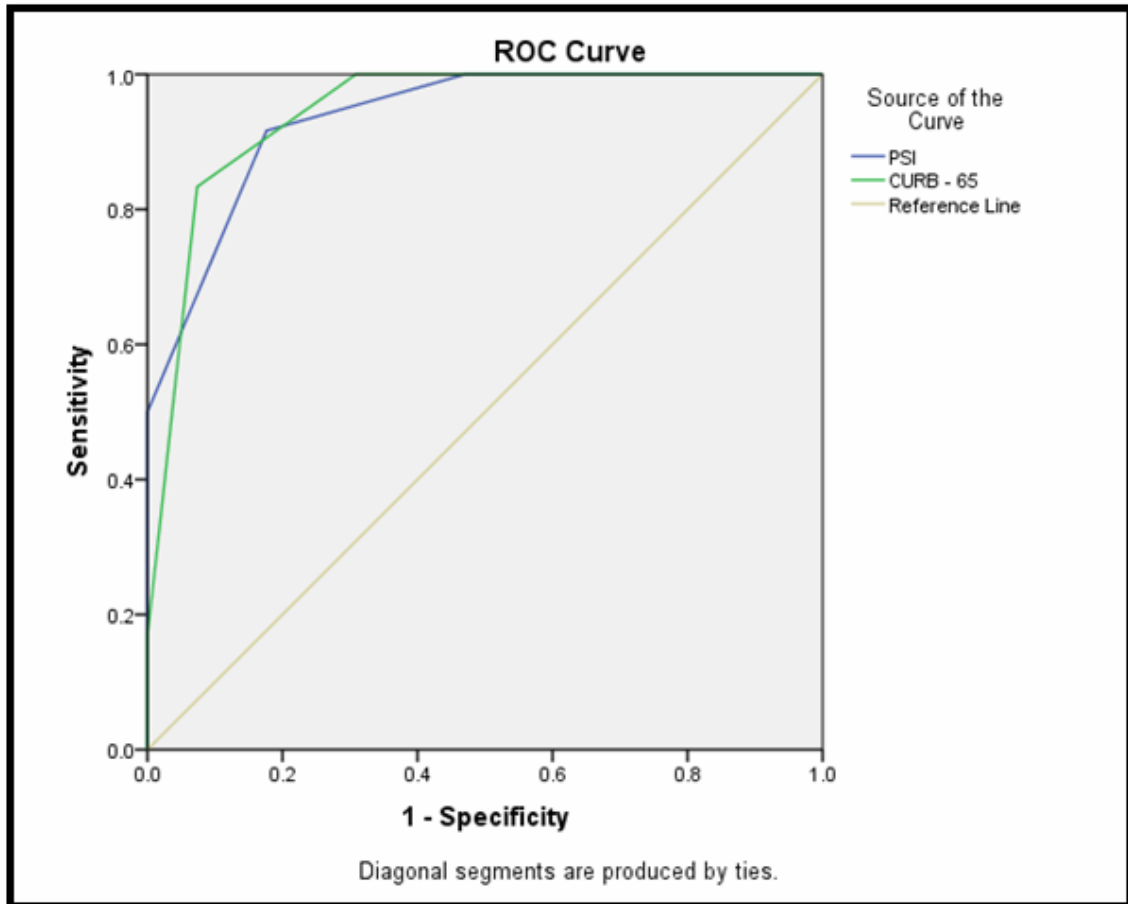


Test Result	Optimum	AUC	Sensitivity	Specificity	P value	95% CI	
Variable(s)	Point of Cut off					Lower Bound	Upper Bound
<b>PSI</b>	>3	0.892	<b>0.962</b>	<b>0.648</b>	<0.0001	0.813	0.972
<b>CURB - 65</b>	>2	0.781	<b>0.731</b>	<b>0.741</b>	<0.0001	0.671	0.892

**Table 28: Sensitivity, Specificity and Area under Curve (AUC) of PSI and CURB65 for Need for ventilation.**

In the study 26 subjects were on ventilator subject. Area under the curve for Ventilator Support patients was highest for PSI score than CURB-65. I.e. PSI score has higher prediction for Ventilator requirement in CAP patients. The p value obtained is within the parameters of significance and hence the association with both the CURB-65 score and PSI score with the Need for Ventilatory Support is significant.

## Prediction of Mortality



**Graph 21: ROC Curve – Comparison of Mortality**

Test	Result	Optimal	Sensitivity	Specificity	Area	P value	95% CI	
Variable(s)		Cut off			Under the Curve		Lower Bound	Upper Bound
<b>PSI</b>		>3	0.917	0.176	0.936	<0.0001	0.873	1.000
<b>CURB - 65</b>		>3	0.833	0.074	0.944	<0.0001	0.890	0.997

**Table 29: Sensitivity, Specificity and Area under Curve (AUC) of PSI and CURB65 for Prediction of Mortality**

In the study 12 subjects had mortality. Area under the curve for Mortality among CAP subjects was highest for CURB-65 score than PSI Score. I.e. CURB Score has higher prediction for Mortality in CAP patients. The p value obtained is within the parameters of significance and hence the association with both the CURB-65 score and PSI score with the Mortality Prediction is significant.

## **DISCUSSION**

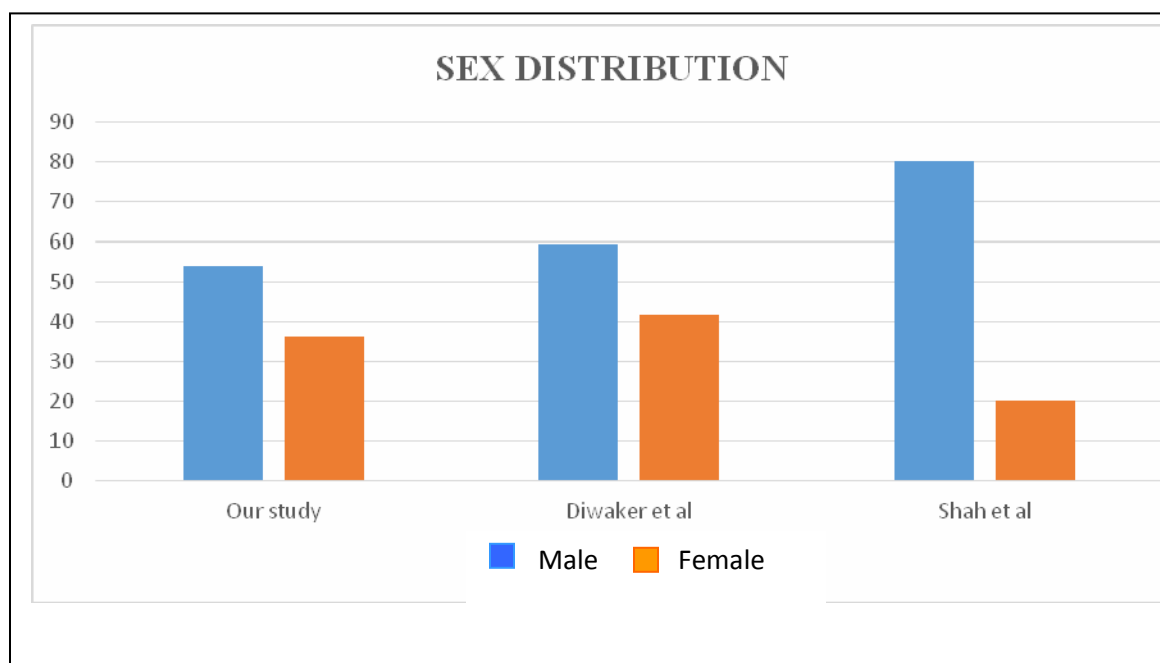
Severe pneumonia remains difficult to define, regardless of the reference used when validating defined criteria. This is mainly due to structural differences across treatment settings with regard to the relative role of emergency departments, intermediate care facilities and ICUs, and ongoing changes in medical practice such as non-invasive ventilation which inherently modify concepts of severity.<sup>21</sup>

Although there are strong similarities between these two methods at first glance, important differences make them unique. PSI uses a long list of predicting factors and its implementation needs various clinical and paraclinical information while CURB-65 is designed to be as simple as possible using a limited set of information. Based on the nature of these two tools, their predictive value largely depends on the environment in which they are implemented. In a hospital setting in a developing countries like ours with scarce resources, simple methods such as CURB-65 are preferred as they put less pressure on the country.

In our study group majority of patients were middle aged and aged 30-60years (59.9%).The largest age group was between 50-60 years. In the study of Dey et al<sup>83</sup>& others they have found out that patients aged > 50 years are more as compared to less than 50 years. It is well documented that pneumonia is commonly occurring disease in the community & its incidence rises sharply with extremes of age. In our study 55% patients were above 50 yrs and 45% patients were below 50 yrs, in study done by Dey et

al 659% patients were above 50yrs and 40% were below 50yrs. Thus pattern of age distribution was comparable.

In our study, there were 43 (54%) male patients and 37 (36%) female patients. In a study done by Metley et al<sup>84</sup> 80% were males and 20% were females. In a study done by Shah BA et al<sup>21</sup> (n=150), 89 (59.3%) were males. This could be attributed to the well-established fact that cigarette smoking and alcoholism, as well as underlying lung disease e.g. COPD predispose to pneumonia and are more common in male population.



**Graph 22: Inter - Study variation of Sex Distribution**

This comparison thus shows a variable distribution of cases but males being more commonly affected than females across all the studies with our study showing a similar pattern.

According to other studies, the mortality risk and the need for ICU admission were higher as the scores increased in both PSI and CURB-65.<sup>4,7</sup> Our study, similarly, revealed that the mortality increased with age, presence of underlying heart failure, high blood levels of urea, pH lower than 7.35, and decreased consciousness level. The most common underlying condition in this study apart from diabetes (23.7%) and hypertension (25%) was heart failure (5%), which had a statistically significant relation with mortality. Also 2.5% of the patients had CKD and 1.3% had Cor pulmonale, Bronchial asthma, Pulmonary TB and post-splenectomy status respectively. Musher et al. in a study on 170 patients with community acquired pneumonia found heart conditions namely CHF in 33 (19.7%) of the patients.<sup>13</sup> Corroborating these results, Lichman et al. reported that 6.8% of their patients had severe heart diseases.<sup>14</sup> In a study done by Shah BA et al<sup>21</sup> 89 patients had one or more co-morbidities. The most common co morbidity was hypertension, followed by diabetes mellitus and chronic obstructive pulmonary disease (COPD).

Smoking history was present in 13.8% of subjects and 2.5% were alcoholic and 10% were both alcoholic and Smokers. In a study done by Shah BA et al<sup>21</sup> eighty-nine patients (59.3%) were smokers of which 74 (83.2%) were males.

In our study among the presenting symptoms 78(97.5%) had cough, making it the most common symptom, among which 91% had associated expectoration and 15% had haemoptysis, 74(92.5%) had fever, 57(71.2%) had breathlessness and 24(30%) had

altered sensorium. In Mac Fartane<sup>85</sup> study of aetiology and outcome of CAP, cough was the most frequent symptom. The other symptoms were fever 86%, chest pain 62% and haemoptysis 15%.

In our study among 80 study population majority of them had total count >11000/microL, which is 67 patients (83.7%). In a study done by Joshua and Michael et al<sup>84</sup> 58% patients had leucocytosis.

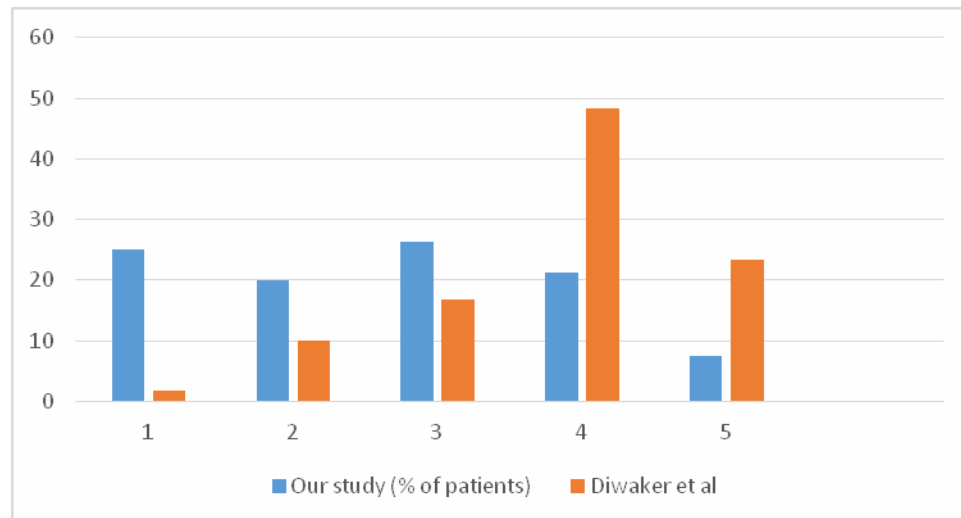
In our study hyponatremia (serum sodium concentration  $\leq$  130 mmol/l) was seen in 23(28.75%) of the patients. In a study done by Dhawan A<sup>86</sup> hyponatraemia was found in 31% of patients at the time of admission, the probable cause of which in 94% of those cases was postulated to be the syndrome of inappropriate antidiuretic hormone secretion (SIADH) in the study.

Use of inotropes and transfusion of blood and blood products was higher in the patients with fatal outcome compared to recovered patients in our study and use of inotropes ( $p < 0.05$ ) showed statistical significance between deaths and recovered and is similar to study done by Gong et al.<sup>87</sup> Use of blood transfusion and its products is not statistically significant between the deaths and recovered in our study. However study done by Gong et al,<sup>87</sup> showed that use of blood and blood transfusion products was significantly high in death patients.

In our study 25% of subjects had 1 score on the Pneumonia Severity Index, 20% had score 2, 26.3% had score 3, 21.3% had score 4 and 7.5% had score 5.. Comparing the data with other studies:

<b>PSI CLASS</b>	<b>Present Study (% of patients)</b>	<b>Diwaker et al (% of patients)</b>
<b>PSI Class 1</b>	<b>25</b>	1.7
<b>PSI Class 2</b>	<b>20</b>	10.0
<b>PSI Class 3</b>	<b>26.3</b>	16.7
<b>PSI Class 4</b>	<b>21.3</b>	48.3
<b>PSI Class 5</b>	<b>7.5</b>	23.3
<b>Total</b>	<b>100</b>	100.0

**Table 30 : Comparison of Distribution of PSI CLASS of patients studied**



**Graph 23: Distribution of Cases - PSI Score**



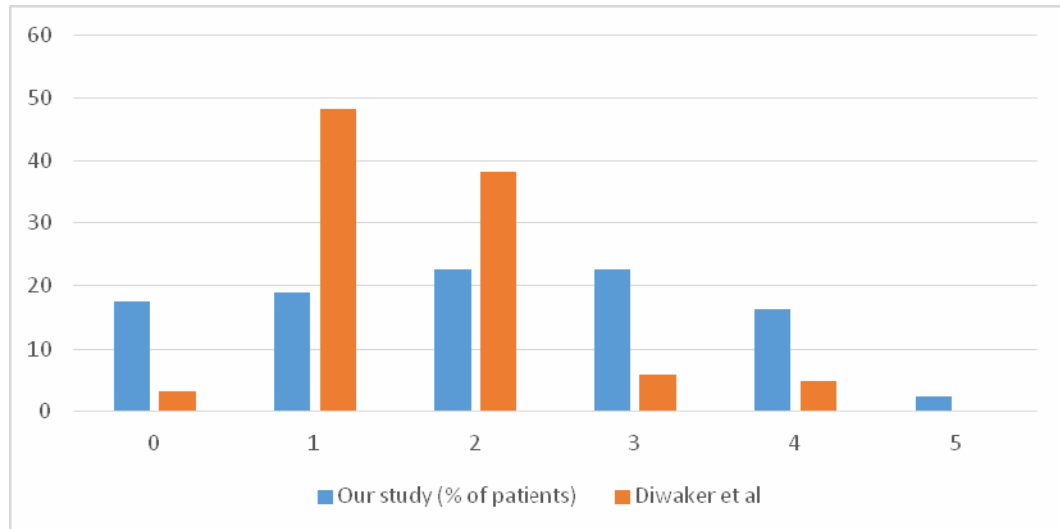
Maximum distribution of cases was seen in PSI Score classes III and IV in our study, while it was in classed IV and V on comparison with Diwaker et al.

When PSI score was compared with various parameters, it was observed that there was significant association between Age, on ventilator support, on inotropes, ICU stay and Mortality. i.e. Higher PSI scores were seen in > 60 years subjects, patients who were put on ventilator, patients on inotropes, patients in ICU and patients who had Mortality. A high degree sensitivity and specificity was observed in PSI score 4 and 5. i.e. higher PSI scores had better diagnostic accuracy in predicting the need for intensive ventilatory and ionotropic support and mortality.

In the study 17.5% had subjects had 0 CURB – 65 score, 18.8% had score 1, 22.5% had score 2 and Score 3 respectively, 16.3% had score 4 and 2.5% had score 5.

**Table 31: Comparison of Distribution of CURB 65score of patients studied**

<b>CURB65 score</b>	<b>PRESENT STUDY (% of patients)</b>	<b>Diwaker et al</b>
<b>CURB65 -0</b>	<b>17.5</b>	3.3
<b>CURB65 -1</b>	<b>18.8</b>	48.3
<b>CURB65 -2</b>	<b>22.5</b>	38.3
<b>CURB65 -3</b>	<b>22.5</b>	6.0
<b>CURB65 -4</b>	<b>16.3</b>	5.0
<b>CURB65 -5</b>	<b>2.5</b>	-
<b>Total</b>	<b>100</b>	100.0



**Graph 24: Distribution of Cases – CURB-65 Score**

Maximum distribution of cases was seen in CURB-65 Score classes I - III in our study, while it was in classed II and III on comparison with Diwaker et al.

Higher sensitivity and specificity was observed in CURB 65 score 4 and 5. I.e. higher CURB -65 scores had better diagnostic accuracy in predicting the need for intensive ventilatory and ionotropic support and mortality.

In a similar study done by Diwaker et al 18 patients had died, 7(24.1%) were in PSI class IV and 6 (42.9%) were in PSI class V and no patients in PSI class 1 died. In 18 mortality patients 8 (34.8%) were in CURB65 class II and only 2 (66.7%) were in class IV and no patients in CURB65 class 0 died. In a study done by Shah BA8 et al sixteen patients (10.7%) died. All the 16 patients (100%) who died were in PSI class >IV. Mortality in PSI class I to III was 0%; in class IV, 14.1% and Class V, 34.8% and in CURB65, class III 2 (12.5%) patients died, class IV 11 (68.7%) patients and class V 3 (18.8%) patients died.<sup>88</sup>

	1		2		3		4		5	
	Count	%	Count	%	Count	%	Count	%	Count	%
<b>PRESENT STUDY</b>	<b>0</b>	<b>0.0%</b>	<b>0</b>	<b>0.0%</b>	<b>1</b>	<b>4.8%</b>	<b>5</b>	<b>29.4%</b>	<b>6</b>	<b>100.0%</b>
<b>Shah et al</b>	0	0.0%	0	0.0%	0	0.0%	8	14%	6	34.7%
<b>Diwaker et al</b>	0	0.0%	1	16.7%	4	40.0%	7	24.1%	6	42.9%

**Table 32: COMPARISON OF MORTALITY AS OUTCOME BY PSI SCORE**

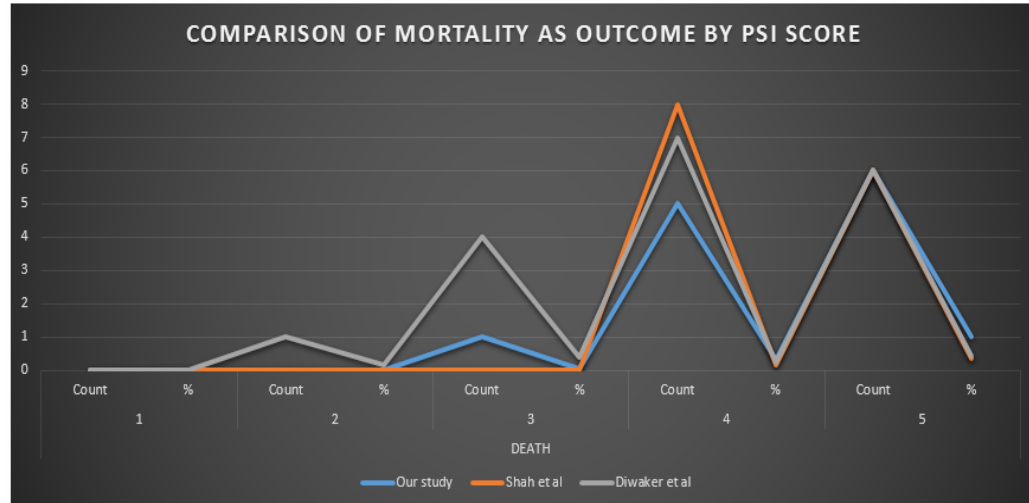


Chart 25: COMPARISON OF MORTALITY AS OUTCOME BY PSI SCORE

	1		2		3		4		5	
	Count	%	Count	%	Count	%	Count	%	Count	%
<b>PRESENT STUDY</b>	0	0.0%	0	0.0%	1	4.8%	5	29.4%	6	100.0%
<b>Diwaker et al</b>	0	0.0%	8	27.6%	8	34.8%	0	0.0%	2	66.7%
<b>Shah et al</b>	0	0.0%	0	0.0%	2	12.5%	11	68.7%	3	18.8%

Table 33: COMPARISON OF MORTALITY AS OUTCOME BY CURB65 SCORE

On comparing our study with those by Diwaker et al and Shah et al it can be inferred that all the 3 study have a similar case distribution and prediction of outcome as Mortality was comparable for various parameters under the various classes of PSI Scoring system (Classes 3-5) and in CURB-65 Scoring system (Classes 3-5).

In the original study by Lim and co-workers of CURB65, mortality risk in the separate groups was as follows: group 1, 3.2%; group 2, 3%; group 3, 17%; group 4, 42%; and group 5, 57 percent <sup>6, 63</sup>. These scores allowed for predictions very similar to those made by the PSI. In a subsequent study,<sup>65</sup> the absence of any CURB criterion was associated with a 30-day mortality of one percent, the presence of one or two with 8%, and the presence of three or four with 30% mortality.

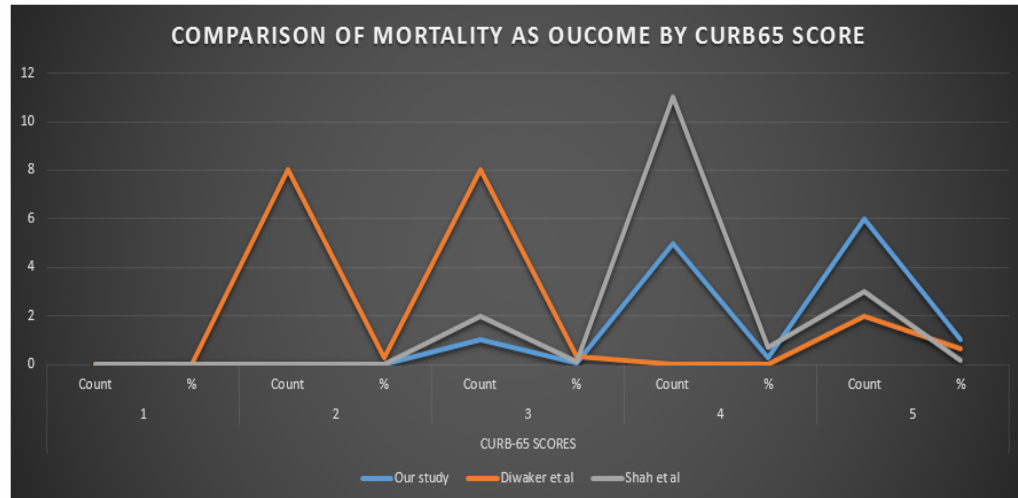
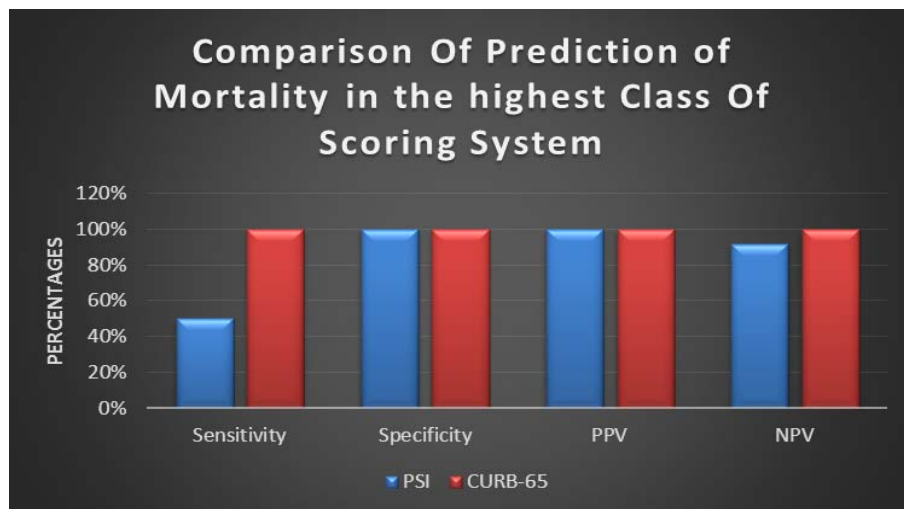


Chart 26: COMPARISON OF MORTALITY AS OUCOME BY CURB-65 SCORE

In the original study by Fine et al of PSI (PORT) mortality rates in risk classes I, II, and III are low (0.1% to 0.4% in class I and 0.9% to 2.8% in class III), with correspondingly higher mortality rates in risk classes IV and V. The cumulative mortality rate of patients in risk classes I to III is less than one percent.



Graph 27: Comparison Of Prediction of Mortality in the highest Class Of the Scoring Systems

In our study on Comparison Of Prediction of Mortality in the highest Class Of Scoring System we have found CURB-65 Scoring system to be the better predictor as it has higher Sensitivity and Negative Predictive Value (NPV) although both the scoring systems have similar Specificity and Positive Predictive Value (PPV).

A significant association between Age, on ventilator support, on inotropes, ICU stay and Mortality was observed with increasing scores in both PSI and CURB-65 scoring systems in our study, with a high degree of sensitivity and specificity.

Thus whereas PSI score has higher prediction for ICU admission and Ventilator requirement in CAP patients, the CURB Score score has higher prediction for Mortality in CAP patients.

In our study in the ROC curve Area under the curve for ICU admitted patients & the patients having received Ventilator Support was higher for PSI score than CURB-65. I.e. PSI score has higher Sensitivity, Specificity and Area under Curve (AUC) but among the 12 subjects who suffered mortality, Area under the curve was highest for CURB-65 score than PSI Score. I.e. CURB Score has higher prediction for Mortality in CAP patients, although PSI Scoring System has higher prediction for ICU admission and need for Ventilatory support. Thus both the scoring systems are complementary to each other.

Capelastegui et al presented a comparative validation of the CURB-65, CRB-65 (which omits the blood urea measurement) and PSI scores in a population of 1,776

patients including 676 outpatients.<sup>89</sup> The 30-day mortality increased with increasing score, and predictions of 30-day mortality were equivalent for all scores as assessed by ROC analysis. This is in contrast to the study by Aujesky et al comprising 3,181 patients and including 1,094 outpatients, showing a minor but significant advantage for the PSI score in predicting 30-day mortality using area under the curve (AUC) analysis.<sup>90</sup> However, this population predominantly included less severely ill patients (only 6% PS IV as compared with 18% in the present study), thereby limiting the comparability of both populations studied.

In line with our results, Shah et al reported both PSI and CURB-65 to have equal sensitivity to predict death from community-acquired pneumonia, adding that PSI was more sensitive in predicting ICU admission than CURB-65.<sup>21</sup> This may be because CURB-65 model does not consider decompensated co-morbidity due to community-acquired pneumonia and results in limited application in the elderly.<sup>70</sup> In another study PSI was reported to have the highest sensitivity followed by CURB-65 in predicting mortality.

The comparison of PSI and CURB-65 with respect to sensitivity, specificity and predictive values have good sensitivity and NPV. These results are comparable to those obtained by Man et al.<sup>70</sup>

Specificity of PSI was found to be better than CURB-65 in contrast to the study by Man et al who postulated their results to the major limitation of the PSI which is the

unbalanced impact of age on the score, resulting in a potential underestimation of severe CAP particularly in younger otherwise healthy individuals.<sup>65</sup>

In another study CURB-65 score of  $\geq 2$  and a PSI score  $>III$  were significantly associated with an increased rate of 28-day mortality, and at a higher percentage per score compared with published data. Compared with the CURB-65, the PSI had a higher sensitivity in predicting mortality and classified a higher proportion of patients as high risk. The PSI was more sensitive than the CURB-65 in predicting 28-day mortality and may serve as a better tool for assessing the risk of pneumonia-related mortality in cancer patients.<sup>91</sup>

The two scoring CURB-65 and PSI approaches are viewed as being complementary, as each has different strengths and weaknesses. The PSI seems to have been developed, and best validated, as a way to identify low mortality risk patients, but the scoring system can occasionally underestimate severity of illness, especially in young patients without comorbid illness.<sup>66, 69</sup> this is primarily because the PSI heavily weighs age and comorbidity, and does not directly measure CAP-specific disease severity.

In contrast, the CURB-65 approach may be ideal for identifying high mortality risk patients with severe illness due to CAP who might otherwise be overlooked without formal assessment of subtle aberrations in key vital signs.<sup>91</sup> However, one clear deficiency of the CURB-65 approach is that it does not generally account for comorbid illness, and thus may not be easily applied in older patients who may still have substantial



mortality risk, even if a mild form of CAP destabilises a chronic, but compensated, disease process. Thus, both tools offer a valuable assessment of patient illness, but from different perspectives, and each is best at identifying patients at opposite ends of the disease severity spectrum.

In a recent studies done by Agrawal et al and Lalitha et al comparing prognostic utility of procalcitonin (PCT) with biomarkers and clinical risk scores (PSI and CURB-65) it was concluded that the management of severe CAP would be greatly improved if it were possible to identify, early in the course of disease, those patients who are most likely to develop complications and are at the risk of mortality with a combined approach of estimating biomarkers and severity scores in collusion.<sup>93, 94</sup>

For predicting ICU admission, however, other indices such as modified ATS, SMART-COP and IDSA/ATS were reported to perform better than PSI and CURB-65, as these indices were originally designed to assess ICU admission rather than mortality.<sup>95</sup> Therefore, a poor performance could be found if applied in predicting mortality.

## **CONCLUSION**

1. The comparison between mortality rates in different risk classes in our study and that of the previous studies showed that in all the studies mortality rates progressively increases with increasing risk scores in both PSI and CURB-65 risk classes.
2. PSI score has higher Sensitivity, Specificity and Area under Curve (AUC) for all the three parameters i.e. prediction of ICU admission in patients, them having received Ventilator Support and probability of Mortality among CAP subjects.
3. Whereas PSI score has higher prediction for ICU admission and Ventilator requirement in CAP patients, the CURB-65 Score has higher prediction for Mortality in CAP patients.
4. A significant association between Age, Requirement of ventilatory support, inotropic support, ICU stay and Mortality was observed with increasing scores in both PSI and CURB-65 scoring systems in our study, with a high degree of sensitivity and specificity.
5. The two scoring CURB-65 and PSI approaches are complementary, as each has different strengths and weaknesses.
6. By using the knowledge of these criteria, patients of CAP can be better prognosticated as regards severity of their illness with consequently better triaging of patients, utilisation of resources and appropriate treatment to improve the outcome in this disease.

7. The use of Biomarkers and Scoring Systems together will improve the predictive power especially in the younger age groups where PSI falters, particularly due to its high dependency on the patient's age.
8. The severity score for community-acquired pneumonia seems to be the preferred method to predict the need for ICU admission and the prognosis of patients seen at Emergency Departments.
9. Despite having comparable specificity and sensitivity with PSI, CURB-65 is much easier to be implemented.
10. Both the scoring systems are applicable and dependable although the ease of use of CURB-65 makes it the scoring system of choice.

## **SUMMARY**

Pneumonia has been considered a health problem for ages. Tools that objectively assess the risk of adverse outcomes, including severe illness and death, help to identify patients who will benefit from hospital care. The two prominent tools for this purpose are the pneumonia severity index (PSI), developed in the USA after pneumonia outcome research trial (PORT), and the CURB-65 rule developed in the U.K. as “confusion, elevated blood urea nitrogen, elevated respiratory rate, low systolic or diastolic blood pressure (BP), and age over 65 years (CURB-65)” rule.

This was a prospective observational study done at a tertiary medical college hospital. Eighty patients aged 18 years or more, who were diagnosed to be having community-acquired pneumonia admitted in R L Jalappa Hospital satisfying the laid criteria were included in the study. All the patients were assessed using Pneumonia Severity Index scoring and CURB-65 scoring.

A significant association between Age, on ventilator support, on inotropes, ICU stay and Mortality was observed with increasing scores in both PSI and CURB-65 scoring systems in our study, with a high degree of sensitivity and specificity. Whereas PSI score has higher prediction for ICU admission and Ventilator requirement in CAP patients, the CURB-65 Score has higher prediction for Mortality in CAP patients. A similar pattern was noted in ROC curves. Thus both the scoring systems have proved to be complementary to each other.

## **BIBLIOGRAPHY**

1. Gleckman RA, Roth RM. Community acquired bacterial pneumonia in the elderly. *Pharmacotherapy* 1984; 4: 81.
2. Horovitz, E.Manor, and Porath. Community acquired pneumonia;aetiology and usefulness of severity criteria on admission.*Thorax*.1996 Oct;51(10);1010-6
3. Welte, Tobias M D,Kohnlein, Thomas;Seminar in Respiratory & Critical Care Medicine. Community Acquired Pneumonia. April2009;30(2);127-135.
4. Moran GJ, Talan D. Pneumonia. In: Marx JA, Hockberger RS, Walls RM, editors. *Rosen's emergency medicine: concepts and clinical practice*. 7th ed. Philadelphia: Elsevier; 2009; 627-938.
5. Fine MJ, Auble TE, Yealy DM, Hanusa BH, Weissfeld LA, Singer DE, et al. A prediction rule to identify low-risk patients with community-acquired pneumonia. *N Engl J Med*; 1997;336:243-50.
6. Lim WS, van der Erden MM, Laing R, Boersma WG, Karalus N, Town GI, et al. defining community acquired pneumonia severity on presentation to hospital: an international derivation and validation study. *Thorax*; 2003;58:377-82.
7. Seaton D. Pneumonia. In: Seaton A, Seaton D. Leitch AG editors. *Crofton & Douglas's. Respiratory Diseases*.5th edition. London. Blackwell Science; 2000:356-429.
8. Shann F, Gratten M, Germer S et al. Aetiology of pneumonia in children in Goroka Hospital, Papua New Guinea.*Lancet* 1984; ii: 537.
9. Gwatkin DR. Acute respiratory infections in under-fives: 15 million deaths a year. *Lancet* 1985; ii: 699.

10. Tuazon CU, Caroella TA, Sheagren JN. Staphylococcal endocarditis in drug users: clinical and microbiologic aspects. *Arch Intern Med* 1975; 135: 1555.
11. Curwen M. Trends in Respiratory Mortality 1951–1975, England and Wales. London: HMSO, 1981.
12. Garibaldi RA, Britt MR, Coleman ML et al. Risk factors for post-operative pneumonia. *Am J Med* 1981; 70: 677.
13. Stevens RM, Teres D, Skillman JJ, Feingold DS. Pneumonia in an intensive care unit. *Arch Intern Med* 1974; 134: 160.
14. Cassiere HA, Niederman MS. New etiopathogenic concepts of ventilator associated pneumonia. *Semin Respir Infect* 1996; 11: 13.
15. Stratton CW. Bacterial pneumonias: an overview with emphasis on pathogenesis, diagnosis and treatment. *Heart Lung* 1986; 15: 226.
16. British Thoracic Society Research Committee. Community-acquired pneumonia in adults in British hospitals in 1982–83: a BTS/PHLS survey of aetiology, mortality, prognostic factors and outcome. *Q J Med* 1987; 62: 195.
17. Rello J, Torres A. Microbial causes of ventilator-associated pneumonia. *Semin Respir Infect* 1996; 11: 24.
18. Karetzky M. Community-acquired pneumonia. In: Brandstetter RD, Karetzky M, Cunha BA, editors. *The Pneumonias*. New York: Springer-Verlag; 1993: 25-48.
19. Regional situation on health statistics reporting. Health Situation in the South-East Asia Region 1994-1997. New Delhi: EHI/WHO-SEARO. September 2007.

20. Garenne M, Ronsmans C, Campbell H (1992). "The magnitude of mortality from acute respiratory infections in children under 5 years in developing countries". *World Health Statistics Quarterly* 45 (2–3): 180–91.
21. Shah BA, Ahmed W, Dhobi GN, Shah NN, Khursheed SQ, Haq I. Validity of pneumonia severity index and CURB-65 severity scoring systems in community acquired pneumonia in an Indian setting. *Indian J Chest Dis Allied Sci*; 2010;52:9-17.
22. Arms RA, Dines DE, Tinstman TC. Aspiration pneumonia. *Chest* 1974; 65: 136.
23. Winfield JB, Sande MA, Gwaltney JM. Aspiration during sleep. *JAMA* 1973; 223: 1288.
24. Ford GT, Whitelaw WA, Rosenal TW. Diaphragm function after upper abdominal surgery in humans. *Am Rev Respir Dis* 1983; 127: 431.
25. Argov S, Goldstein I, Barzilai A. Is routine use of the nasogastric tube justified in upper abdominal surgery? *Am J Surg* 1980; 139: 849.
26. Smith FE, Palmer DL. Alcoholism, infection and altered host defenses. A review of clinical and experimental observations *J Chron Dis* 1976; 29: 35.
27. Craven DE, Lichtenberg DA, Goularte TA et al. Contaminated medication nebulizers in mechanical ventilator circuits: source of bacterial aerosols. *Am J Med* 1984; 77: 834.
28. Centers for Disease Control. Recommendations for the disinfection and maintenance of respiratory therapy equipment. Atlanta: Centers for Disease Control, 1980.
29. Haas H, Morris JF, Samson S et al. Bacterial flora of the respiratory tract in chronic bronchitis: comparison of trans-tracheal, fiberbronchoscopic and oropharyngeal sampling methods. *Am Rev Respir Dis* 1977; 116: 41.
30. Tillotson JR, Lernler AM. Pneumonia caused by Gram-negative bacilli. *Medicine* 1966; 45:65.

31. Tillotson JR, Lerner AM. Characteristics of pneumonia caused by *Escherichia coli*. *N Engl J Med* 1967; 227: 115.
32. Fisher AM, Trever RW, Curtin JA et al. Staphylococcal pneumonia: a review of 21 cases adults. *N Engl J Med* 1958; 258: 919.
33. Levi J, Robson M, Rosenfeld JB. Septicaemia and pulmonary embolism complicating use of arteriovenous fistula in maintenance haemodialysis. *Lancet* 1970; ii: 288.
34. Li JZ - Efficacy of short-course antibiotic regimens for community acquired pneumonia: a meta-analysis *Am J Med* - 01-SEP-2007; 120(9): 783-90.
35. Lieberman, D., F. Schlaeffer, I. Boldur, D. Lieberman, S. Horowitz, M. G. Friedman, M. Leiononen, O. Horovitz, E. Manor, and A. Porath. Multiple pathogens in adult patients admitted with community-acquired pneumonia. A one year prospective study of 346 consecutive patients. *Thorax* 1996;51; 179-184.
36. Neill AM, Martin IR, Weir R, et al. Community acquired pneumonia: aetiology and usefulness of severity criteria on admission. *Thorax*. 1996;51(10):1010-1016.
37. Baselki US, Wunderink RG. Bronchoscopic diagnosis of pneumonia. *Clin Microbiol Rev* 1994; 7: 533.
38. Garenne M, Ronsmans C, Campbell H (1992). "The magnitude of mortality from acute respiratory infections in children under 5 years in developing countries". *World Health Statistics Quarterly* 45 (2-3): 180-91.
39. Seaton A. Other pulmonary neoplasm and related conditions. Crofton and Douglas's Respiratory Diseases. In: Seaton A, Seaton D, Leitch AG, editors. 5 th ed. Oxford: Blackwell science ltd; 2000.



40. Boersma WG, Daniels JM, Löwenberg A, Boeve WJ, van de Jagt EJ. Reliability of radiographic findings and the relation to etiologic agents in community-acquired pneumonia. *Respir Med.* 2006 May. 100(5):926-32.
41. American Thoracic Society. 1993. Guidelines for the initial management of adults with community-acquired pneumonia: diagnosis, assessment of severity, and initial antimicrobial therapy. *Am. Rev. Respir. Dis.* 148: 1418-1426.
42. Murray, T. J., and J. A. Washington. 1975. Microscopic and bacteriologic analysis of expectorated sputum. *Mayo Clin. Proc.* 50: 339-344.
43. American Thoracic Society/Infectious Diseases Society of America: Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare associated pneumonia. *Am J Respir Crit Care Med* 171:388, 2005.
44. Chalasani NP, Valdecanas MAL, Gopal AK. Clinical utility of blood cultures in adult patients with community-acquired pneumonia without defined underlying risks. *Chest* 1995;108:932-6.
45. Richard G. Wunderink and Grant W. Waterer Appropriate Microbiological Testing in Community-Acquired Pneumonia *Chest* 2001;119; 5-7.
46. Jay SJ, Johanson WG, Pierce AK. The radiographic resolution of *Streptococcus pneumoniae* pneumonia. *N Engl J Med* 1975; 293: 798.
47. Woodhead MA, Macfarlane JT, McCracken JS et al. Prospective study of the etiology and outcome of pneumonia in the community. *Lancet* 1987; i: 671.
48. Parrino TA, Stollerman GH. The management of pneumonia. *Adv Intern Med* 1984;0:113.

49. Hirani NA, Macfarlane JT. Impact of management guidelines on the outcome of severe community acquired pneumonia. *Thorax*; 1997;52:17-21.
50. British Thoracic Society Research Committee and Public Health Laboratory Service. The aetiology, management and outcome of severe community-acquired pneumonia on the intensive care unit. *Respir Med*;1992;86:7-13.
51. Chalfin DB, Trzeciak S, Likourezos A, Baumann BM, Dellinger RP: Impact of delayed transfer of critically ill patients from the emergency department to the intensive care unit. *Crit Care Med* 2007, 35:1477-1483.
52. Restrepo MI, Mortensen EM, Velez JA, Frei C, Anzueto A: A comparative study of community-acquired pneumonia patients admitted to the ward and the ICU. *Chest* 2008, 133:610-617.
53. Renaud B, Santin A, Coma E, Camus N, Van Pelt D, Hayon J, et al.: Association between timing of intensive care unit admission and outcomes for emergency department patients with community-acquired pneumonia. *Crit Care Med* 2009, 37:2867-2874.
54. Carron M, Freo U, Zorzi M, Ori C. Predictors of failure of noninvasive ventilation in patients with severe community-acquired pneumonia. *J Crit Care*. 2010 Sep. 25(3):540.e9-14.
55. Aujesky D, Auble TE, Yealy DM et al. Prospective comparison of three validated prediction rules for prognosis in community-acquired pneumonia. *Am. J. Med*;2005; 118: 384–92.
56. Guest JF, Morris A. Community-acquired pneumonia: the annual cost to the National Health Service in the United Kingdom. *Eur Respir J* 1997;10:1530-4.

57. Fang GD, Fine M, Orloff J, Arisumi D, Yu VL, Kapoor W, et al. New and emerging etiologies for community-acquired pneumonia with implications for therapy: a prospective multicenter study of 359 cases. *Medicine* 1990;69:307-16.
58. Ortqvist A, Hedlund J, Grillner L, Jalonon E, Kallings I, Leinonen M, et al. Aetiology outcome and prognostic factors in community acquired pneumonia requiring hospitalization. *Eur Respir J* 1990;3:1105-13.
59. Anonymous. Community-acquired pneumonia in adults in British hospitals in 1982-1983: a survey of aetiology, mortality, prognostic factors and outcome. The British Thoracic Society and the Public Health Laboratory Service. *Q J Med* 1987; 62:195-220.
60. Armitage P. Statistical methods in medical research. Oxford:Blackwell Scientific Publications, 1971.
61. Macfarlane JT. Adverse prognostic factors in pneumonia. *Thorax* 1983;38:231.
62. Lim WS, Lewis S, Macfarlane JT. Severity prediction rules in community-acquired pneumonia: a validation study. *Thorax* 2000;55:219-23.
63. British Thoracic Society Standards of Care Committee. BTS Guidelines for the Management of Community Acquired Pneumonia in Adults. *Thorax* ;2001;56: 1–64.
64. Ewig S, de Roux A, Garcia E, Mensa J, Niederman M, Torres A. Validation of predictive rules and indices of severity for community-acquired pneumonia. *Thorax* 2004;59:421-7.
65. Ewig S, Kleinfeld T, Bauer T, Seifert K, Schäfer H, Göke N. Comparative validation of prognostic rules for community acquired pneumonia in an elderly population. *Eur Respir J* 1999;14:370-5.

66. Roson B, Carratala J, Dorca J, Casanova A, Manresa F, Gudiol F. Etiology, reasons for hospitalization, risk classes, and outcomes of community-acquired pneumonia in patients hospitalized on the basis of conventional admission criteria. *Clin Infect Dis* 2001;33:158-65.
67. Mortensen EM, Kapoor WN, Chang CCH, Fine MJ. Assessment of mortality after long-term follow-up of patients with community-acquired pneumonia. *Clin Infect Dis* 2003;37:1617-24.
68. Buising KL, Thursky KA, Black JF, MacGregor L, Street AC, Kennedy MP, et al. A prospective comparison of severity scores for identifying patients with severe community acquired pneumonia: reconsidering what is meant by severe pneumonia. *Thorax* 2006;61:419-24.
69. Man SY, Lee N, Ip M, Antonio GE, Chau SS, Mak P, et al. Prospective comparison of three predictive rules for assessing severity of community-acquired pneumonia in Hong Kong. *Thorax* 2007;62:348-53
70. Niederman MS, Mandell LA, Anzueto A, Bass JB, Broughton WA, Campbell GD, Dean N, File T, Fine MJ, Gross PA, et al. Guidelines for the management of adults with community-acquired pneumonia: diagnosis, assessment of severity, antimicrobial therapy, and prevention. *Am J Respir Crit Care Med* 2001;163:1730–1754.
71. Eurich DT, Sadowski CA, Simpson SH, Marrie TJ, Majumdar SR. Recurrent community-acquired pneumonia in patients starting acid-suppressing drugs. *Am J Med.* 2010 Jan. 123(1):47-53.

72. Singh N et al: Short-course empiric antibiotic therapy for patients with pulmonary infiltrates in the intensive care unit. A proposed solution for indiscriminate antibiotic prescription. *Am J Respir Crit Care Med* 2000;162:505.
73. ERS Task Force Report. Guidelines for management for adult community acquired lower respiratory tract infections. *Eur Respir J* 1998, 11 : 986-91.
74. Boulware DR, Daley CL, Merrifield C, Hopewell PC, Janoff EN. Rapid diagnosis of pneumococcal pneumonia among HIV-infected adults with urine antigen detection. *J Infect.* 2007 Oct. 55(4):300-9.
75. Fagon JY et al: Invasive and noninvasive strategies for management of suspected ventilator-associated pneumonia. A randomized trial. *Ann Intern Med* 132:621, 2000.
76. Adnet F, Baud F. Relation between Glasgow Coma Scale and aspiration pneumonia. *Lancet.* 1996 Jul 13. 348(9020):123-4.
77. The British Thoracic Society. Guidelines for the management of community-acquired pneumonia in adults admitted to hospital. *Br J Hosp Med* 1993;49:346-350.
78. Johnstone J, Marrie TJ, Eurich DT, Majumdar SR. Effect of pneumococcal vaccination in hospitalized adults with community-acquired pneumonia. *Arch Intern Med.* 2007 Oct 8. 167(18):1938-43.
79. Centers for Disease Control and Prevention. Use of 13-Valent Pneumococcal Conjugate Vaccine and 23-Valent Pneumococcal Polysaccharide Vaccine for Adults with Immunocompromising Conditions: Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 2012 Oct 12. 61:816-9.
80. Tomczyk S, Bennett NM, Stoecker C, Gierke R, Moore MR, Whitney CG, et al. Use of 13-Valent Pneumococcal Conjugate Vaccine and 23-Valent Pneumococcal

Polysaccharide Vaccine Among Adults Aged  $\geq 65$  Years: Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Morb Mortal Wkly Rep. 2014 Sep 19. 63(37):822-5.

81. Bonten M, Bolkenbaas M, Huijts S, et al. Community Acquired Pneumonia Immunization Trial in Adults (CAPiTA). Abstract no. 0541. Pneumonia 2014;3:95.
82. Dey et al. Clinical presentation and predictors of outcome in adult patients with community-acquired pneumonia. Natl Med-India. 1997 July-Aug; 104: 169-172.
83. Metlay J P, Fine M J. Testing strategies in the initial management of patients with community-acquired pneumonia. Ann Intern Med. 2003 Jan 21;138(2):109-18. Review.
84. Mac farlane JT, Finch RG, Ward MJ, Macrae AD. Hospital study of adult community acquired pneumonia. Lancet. 1982 Jul 31;2(8292):255-8.
85. Dhawan A, Narang A, Singhi S. Hyponatraemia and the inappropriate ADH syndrome in pneumonia. Annals of Tropical Paediatrics 1992;12:455-62.
86. Gong MNB, Thompson T, Williams P, Pothier L, Boyee PD, Christiani DC. Clinical predictors of and mortality in acute respiratory distress syndrome: Potential role of red cell transfusion. Crit Care Med 2005;33(6):1191-1198.
87. Diwakar T. N, Ravish K. S, Zakir Hussain G. Comparative Study Of Pneumonia Severity Index And Curb65 In Assesing The Severity Of Community Acquired Pneumonia. Journ of med and dental sciences 2013; FEB. 836-850.
88. Capelastegui A, Espana PP, Quintana JM, Areitio I, Gorordo I, Egurrola M, et al. Validation of a predictive rule for the managment of community-acquired pneumonia. Eur Respir J 2006;27:151-7.

89. Aujesky D, Auble TE, Yealy DM, Stone RA, Obrosky DS, Meehan TP, et al. Prospective validation of three validated prediction rules for prognosis in community-acquired pneumonia. *Am J Med* 2005;118:384-92.
90. Gonzalez, Carmen, et al. "Predicting pneumonia mortality using CURB-65, PSI, and patient characteristics in patients presenting to the emergency department of a comprehensive cancer center." *Cancer medicine* 3.4 (2014): 962-970.
91. Roson B, Carratala J, Dorca J, Casanova A, Manresa F, Gudiol F. Etiology, reasons for hospitalization, risk classes, and outcomes of community-acquired pneumonia in patients hospitalized on the basis of conventional admission criteria. *Clin Infect Dis* 2001;33:158-65.
92. Agarwal SK, Meena M, Misra AK, Meena LP, Singh M. A study to compare prognostic utility of procalcitonin with existing biomarkers (CRP and TLC) and clinical risk scores (PSI and CURB 65) in community acquired pneumonia. *Natl J Physiol Pharm Pharmacol*. 2015; 5(1): 28-32.
93. Lalita Fernandes, Akashdeep Singh Arora, Anthony Menezes Mesquita. Role Of Semi-Quantitative Serum Procalcitonin In Assessing Prognosis Of Community Acquired Bacterial Pneumonia Compared To Port Psi, Curb-65 And Crb-65. *Journal of Clinical and Diagnostic Research*; Jul,2015; 1041-1052.
94. Fang WF, Yang KY, Wu CL, et al. Application and comparison of scoring indices to predict outcomes in patients with healthcare associated pneumonia. *Crit Care*. 2011;15:R32.

## **ANNEXURES**

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH**

**TAMAKA, KOLAR-563101**

***Informed Consent Form for Clinical Studies***

**Informed Consent form for “Comparison of Severity Scoring Systems in Predicting the Prognosis of Community Acquired Pneumonia.”**

This Informed Consent Form is for men and women who attend the outpatient and inpatient at R.L.Jalappa Hospital and who we are inviting to participate in research on **Community Acquired Pneumonia.**

The title of our research project is **“Comparison of Severity Scoring Systems in Predicting the Prognosis of Community Acquired Pneumonia.”**

The study will be conducted by Dr. Uphar Gupta under the guidance of Dr. Vidyasagar CR from the department of General Medicine.



**This Informed Consent Form has two parts:**

- **Information Sheet (to share information about the research with you)**
- **Certificate of Consent (for signatures if you agree to take part)**

**You will be given a copy of the full Informed Consent Form**

## **PART I: Information Sheet**

### **Introduction**

I am Dr. Uphar Gupta, working under the guidance of Dr. Vidyasagar CR from R.L.Jalappa Research Institute. We are doing research on Community Acquired Pneumonia , which is very common in this country. I am going to give you information and invite you to be part of this research. You do not have to decide today whether or not you will participate in the research. Before you decide, you can talk to anyone you feel comfortable with about the research.

There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have questions later, you can ask them of me, the study doctor or the staff.

### **Purpose of the research**

Pneumonia is defined as an acute inflammation of the pulmonary parenchyma that can be caused by various infective and non-infective agents, presenting with physical and radiological features compatible with pulmonary consolidation of a part or parts of one or both lungs the scoring systems currently employed in the western world have not been

validated in developing countries where population demographics and health-care delivery systems are different from the developed world.

The condition imposes a heavy burden on the healthcare system in terms of its high cost both for diagnosing and treating the condition as well as for the hospital and ICU stay. This heavy cost points out the importance of predicting the need for hospitalization as well as the outcome of these patients. Prognostic scoring systems for CAP have been developed to address these issues.

Even though most of the burden in terms of mortality and morbidity occurs in the developing world, little has been done to study the factors associated with an adverse prognosis in CAP in this region. The purpose of the study is to test the validity of PSI and CURB-65 severity scoring systems in CAP in predicting outcome and need for ICU admissions in patients coming to R L Jalappa Hospital, a tertiary care centre.

### **Type of Research Intervention**

This research will involve collection of clinical history and necessary investigations

such as chest radiograph (postero-anterior or antero-posterior views) at presentation; electrocardiogram; arterial blood gas analysis and serum electrolyte measurement; sputum for gram staining and culture; complete blood counts, blood urea nitrogen and serum creatinine; fasting blood glucose.

**Participant selection**

We are inviting all individuals who attend outpatients and inpatients to participate in the research on study of Community Acquired Pneumonia.

**Voluntary Participation**

Your participation in this research is entirely voluntary. It is your choice whether to participate or not. Whether you choose to participate or not, all the services you receive at this clinic will continue and nothing will change. If you choose not to participate in this research project, you will offered the treatment that is routinely offered in this clinic/hospital for rheumatoid arthritis, and we will tell you more about it later. You may change your mind later and stop participating even if you agreed earlier.

**Procedures and Protocol**

We will be taking history , performing clinical examination and nescessarey investigations such as chest radiograph (postero-anterior or antero-posterior views) at presentation; electrocardiogram; arterial blood gas analysis and serum electrolyte measurement; sputum for gram staining and culture; complete blood counts, blood urea nitrogen and serum creatinine; fasting blood glucose.

**Duration**

The research takes place over 6 wks from the day of admission to the hospital.

**Side Effects**

No side effects in participating in the study.

**Risks:**

NO SIGNIFICANT RISK INVOLVED FOR PARTICIPATING IN THE STUDY.

**Benefits**

If you participate in this research, you will have the following benefits: any interim illnesses will be treated at no charge to you. There may not be any benefit for you but your participation is likely to help us find the answer to the research question. There may not be any benefit to the society at this stage of the research, but future generations are likely to benefit.

**Reimbursements**

You will not be given any money or gifts to take part in this research.

**Confidentiality**

With this research, something out of the ordinary is being done in your community. It is possible that if others in the community are aware that you are participating, they may ask you questions. We will not be sharing the identity of those participating in the research.

The information that we collect from this research project will be kept confidential. Information about you that will be collected during the research will be put away and no one but the researchers will be able to see it. Any information about you will have a number on it instead of your name. Only the researchers will know what your number is and we will lock that information up with a lock and key. It will not be shared with or given to anyone except Dr. Vidyasagar CR.

**Sharing the Results**

The knowledge that we get from doing this research will be shared with you through community meetings before it is made widely available to the public. Confidential information will not be shared. After these meetings, we will publish the results in order that other interested people may learn from our research.

**Right to Refuse or Withdraw**

You do not have to take part in this research if you do not wish to do so and refusing to participate will not affect your treatment at this clinic in any way. You will still have all the benefits that you would otherwise have at this clinic. You may stop participating in the research at any time that you wish without losing any of your rights as a patient here. Your treatment at this clinic will not be affected in any way.

**Alternatives to Participating**

If you do not wish to take part in the research, you will be provided with the established standard treatment available at the centre/institute/hospital.

### **Who to Contact**

If you have any questions you may ask them now or later, even after the study has started. If you wish to ask questions later, you may contact :

Dr. Uphar Gupta

Postgraduate in general medicine

Sri Devaraj Urs Medical College.

Tamaka Kolar, Karnataka , Pin:563101

Ph no-8971171098 ,Email.id- druphar@gmail.com

**This proposal has been reviewed and approved by Ethical Clearance Committee, which is a committee whose task it is to make sure that research participants are protected from harm. If you wish to find about more about the IRB. It has also been reviewed by the Ethics Review Committee of Sri Devaraj Urs Medical College, which is supporting the study.**

**PART II: Certificate of Consent**

**I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research.**

**Print Name of Participant**\_\_\_\_\_

**Signature of Participant** \_\_\_\_\_

**Date** \_\_\_\_\_

**Day/month/year**

**If illiterate**

**I have witnessed the accurate reading of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.**

**Print name of witness**\_\_\_\_\_

**AND**

**Thumb print of**

**participant**

**Signature of witness** \_\_\_\_\_

**Date** \_\_\_\_\_

**Day/month/year**



**Statement by the researcher/person taking consent**

**I have accurately read out the information sheet to the potential participant, and to the best of my ability made sure that the participant understands that the following will be done:**

**1.**

**2.**

**3.**

**I confirm that the participant was given an opportunity to ask questions about the study, and all the questions asked by the participant have been answered correctly and to the best of my ability. I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.**

**A copy of this ICF has been provided to the participant.**

**Print Name of Researcher/person taking the consent**\_\_\_\_\_

**Signature of Researcher /person taking the consent**\_\_\_\_\_

**Date** \_\_\_\_\_ **(Day/month/year)**



ಮಾಹಿತಿಯುಕ್ತ ಸಮ್ಮತಿಯ ನಮೂನೆ ಸಮೂಹ ಕಠೋರತೆಯ ಅಂಕ ವಿಧಾನಗಳು  
ಪೂರ್ವಸೂಚಕ ಮೌಲ್ಯದ ಹೋಲಿಕೆ ಮೋನಿಯಾ ಸ್ವಾಧೀನ ಪಡಿಸಿಕೊಂಡಿತು

ಸಮುದಾಯ ಸ್ವಾಧೀನಪಡಿಸಿಕೊಂಡಿತು ನ್ಯೂಮೋನಿಯಾ (ಸಿಎಪಿ) ಅಭಿವೃದ್ಧಿಶೀಲ ದೇಶಗಳಲ್ಲಿ 30 % ರಿಂದ 20 % ರಷ್ಟು ಪ್ರಕರಣಗಳಲ್ಲಿ ಸಾಮಾನ್ಯ ವ್ಯಾಧಿ . ಇದು ಸೂಕ್ತ ಪೂರ್ವಸೂಚಕ ಅಂಶಗಳಲ್ಲಿ ಜ್ಞಾನ ತೀವ್ರ ನಿಗಾ ಚಿಕಿತ್ಸೆಯ ಅಗತ್ಯ ಹೆಚ್ಚಿನ ಅಪಾಯ ರೋಗಿಗಳ ಆರಂಭಿಕ ಗುರುತಿನ ಉಪಯುಕ್ತ ಇರಬಹುದು ಭರವಸೆಯಿದೆ . ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪುತ್ತೀರಿ ವೇಳೆ ನೀವು ಅಥವಾ ನೀವು ಅಥವಾ ಎರಡೂ ಜವಾಬ್ದಾರಿ ವ್ಯಕ್ತಿಯಿಂದ ಮಾಹಿತಿ (ಪ್ರತಿ proforma ಮಾಹಿತಿ ) ಸಂಗ್ರಹಿಸುತ್ತದೆ . ನಿಮ್ಮ ಆಸ್ಪತ್ರೆ ದಾಖಲೆಯಿಂದ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಸೂಕ್ತ ವಿವರಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತದೆ . ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿ ಮಾತ್ರ ಪ್ರೌಢಪ್ರಬಂಧದಲ್ಲಿ ಮತ್ತು ಪ್ರಕಟಣೆ ಬಳಸಲಾಗುತ್ತದೆ . ಈ ಅಧ್ಯಯನವು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ವಿಮರ್ಶಿಸುತ್ತದೆ ಮಾಡಲಾಗಿದೆ . ನೀವು ಭಾಗವಹಿಸಲು ಇಚ್ಛಿಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುತ್ತಾನೆ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ . ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿಕೊಂಡಲ್ಲಿ ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು ಸೈನ್ / ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ

ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಂತೆ ಮತ್ತು ಈ ನನ್ನ ಮುಂದಿನ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ ಉಚಿತ ಉಳಿಯಲು ಎಂದು ಅರ್ಥ . ನಾನು ಓದಲು ಅಥವಾ ನನಗೆ ಓದಲು ಮಾಡಲಾಗಿದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶ , ಬಳಸಲಾಗುವ ವಿಧಾನ , ಅಧ್ಯಯನ ಮತ್ತು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿದ ಮತ್ತು ಬಹಿರಂಗ ನಡೆಯಲಿದೆ ಮಾಹಿತಿಯನ್ನು ಪ್ರಕೃತಿಯಲ್ಲಿ ನನ್ನ ಒಳಗೊಳ್ಳುವಿಕೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯ ಮತ್ತು ಲಾಭಗಳನ್ನು ಅರ್ಥ . ನಾನು ಅಧ್ಯಯನ ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ವಿವಿಧ ಅಂಶಗಳನ್ನು ನನ್ನ ತೃಪ್ತಿ ಉತ್ತರಿಸುವ ಬಗ್ಗೆ ನನ್ನ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶ ಹೊಂದಿದ್ದರು ನಾನು, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಮತ್ತು ಪ್ರೌಢಪ್ರಬಂಧದಲ್ಲಿ ನನ್ನ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಡಿಸ್ಕ್ಲೋಸರ್ ಅಧಿಕೃತಗೊಳಿಸಲು ಒಪ್ಪುತ್ತೀರಿ ರುಜುಮಾಡಿರುವ

ವಿಷಯ ಹೆಸರು

(ಪಾಲಕರು/ಗಾರ್ಡಿಯನ್ನೇಸರು)

DATE :

ಸಹಿ / ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು

ಒಪ್ಪಿಗೆ ತೆಗೆದುಕೊಳ್ಳುವ ವ್ಯಕ್ತಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

## **PROFORMA**

1. OP/IP No.:
2. Date:
3. Case Serial No.:
4. Name:
5. Age:
6. Gender:
7. Occupation:
8. Date of Admission:
9. Date of Discharge:
10. Socioeconomic status:
11. Address with Phone no.:
12. Chief Complaints:
13. Past history:

14. Family history:

15. Personal History:

16. General Physical Examination: (At admission)

PR:

BP:

Temp:

Resp Rate:

Spo2:

Pallor:

Icterus:

Cyanosis:

Clubbing:

Lymphdenopathy:

Oedema:

17. Systemic examination:

CVS:

RS:

PA:

CNS:

18. Diagnosis:

19. Duration of hospital stay:

20. INVESTIGATIONS:

I. Chest X-ray:

II. Electrocardiogram:

III. Complete Blood Count :

IV. Haematocrit:

V. Arterial Blood Gas Analysis

VI. Blood urea (mg/dl) =

Blood urea nitrogen(mg/dl) = B. urea(mg/dl) / 2.14 =

VII. Serum creatinine

VIII. Serum electrolyte measurement

IX. Fasting blood glucose

X. Serum albumin levels

TOTAL SCORE

1. PSI Score :-

2. CURB-65 Score :-

Signature

Place:

Time:

## **PATIENT INFORMATION SHEET**

### **Comparison of Severity Scoring Systems in Predicting the Prognosis of Community**

#### **Acquired Pneumonia.**

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish.

Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Pneumonia is defined as an acute inflammation of the pulmonary parenchyma that can be caused by various infective and non-infective agents, presenting with physical and radiological features compatible with pulmonary consolidation of a part or parts of one or both lungs; the scoring systems currently employed in the western world have not been validated in developing countries where population demographics and health-care delivery systems are different from the developed world.

The condition imposes a heavy burden on the healthcare system in terms of its high cost both for diagnosing and treating the condition as well as for the hospital and ICU stay. This heavy cost points out the importance of predicting the need for hospitalization as well as the outcome of these patients. Prognostic scoring systems for CAP have been developed to address these issues.

Even though most of the burden in terms of mortality and morbidity occurs in the developing world, little has been done to study the factors associated with an adverse prognosis in CAP in this region. The purpose of the study is to test the validity of PSI and CURB-65 severity scoring systems in CAP in predicting outcome and need for ICU admissions in patients coming to R L Jalappa Hospital, a tertiary care centre.

For the purpose of this study you will have to answer a few simple questions after which physical examination shall be carried out. For the purpose of this study necessary invasive procedures or investigations will be carried out.

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason.

**This will not affect the standard of care you receive.**

### **KEY TO MASTER CHART**

1. CURB – 65 - “confusion, elevated blood urea nitrogen, elevated respiratory rate, low systolic or diastolic blood pressure (BP), and age over 65 years (CURB-65)” rule score.
2. PSI - pneumonia severity index
3. s. K – Serum Potassium
4. s. Na - Serum Sodium
5. s.CREAT - Serum Creatinine
6. BUN – Blood Urea Nitrogen
7. SBP – Systolic Blood Pressure
8. PR – Pulse Rate
9. TEMP – Axillary Temperature
10. R. Rate – Resting Respiratory Rate
11. Y – Presence of the variable
12. N – Absence of the variable
13. UL – Unilobular Involvement
14. ML – Multilobular Involvement



# MASTER CHART

S.No	Hosp.No.	NAME	AGE	SEX	FEVER	ALTERED SENSORIUM	BREATHLESSNESS	COUGH	PAST HISTORY	PERSONAL HISTORY	PR	SBP	TEMP	R. Rate	SpO2	LUNG ABSCESS	CAVITY	EMPHYEMA	P. Effusion	Radiographic features	pH	pO2	pCO2	b.urea	BUN	s.CREAT	s. Na	s. K	s. GLUCOSE	DEATH	VENTILATOR	IONOTROPES	ICU STAY	PSI	CURB - 65
1	970709	SUNITHA	35	F	Y	N	N	Y	N	N	100	120	101	20	100	N	N	N	N	UL	7.38	78	65	23	10.74766355	1	136	3.6	87	N	N	N	N	1	0
2	1004267	NARAYAN SWAMY	52	M	Y	N	Y	Y	N	S	90	100	101	24	94	N	N	N	N	UL	7.4	98	46	11	5.140186916	0.75	126	4.5	87	N	N	N	N	1	0
3	975162	PRABHAVATHI	36	F	Y	N	N	Y	N	N	92	120	98	46	98	N	N	N	N	UL	7.42	89	42	30	14.01869159	0.6	130	2.8	140	N	N	N	N	1	0
4	42445	SRINIVAS	35	M	Y	N	N	Y	N	N	92	120	98	24	98	N	N	N	N	UL	7.34	89	34	12	5.607476636	1.5	134	4.5	106	N	N	N	N	1	0
5	43717	CHIMAPPA	55	M	Y	N	N	Y	N	N	92	130	98	25	98	N	N	N	N	UL	7.36	86	40	39	18.22429907	0.8	136	3.9	112	N	N	N	N	1	0
6	101891	VENKATESHAPPA	45	M	Y	N	N	Y	N	N	97	110	99	26	94	N	N	N	N	UL	7.34	86	34	21	9.813084112	1	134	3.7	132	N	N	N	N	1	0
7	98756	CHOWDAPPA	32	M	Y	N	Y	Y	N	N	96	110	98	28	98	N	N	N	N	UL	7.38	86	30	24	11.21495327	1	132	3.9	117	N	N	N	N	1	0
8	42068	BHAGYAMMA	54	F	Y	N	N	Y	N	N	95	100	100	35	96	N	N	N	N	UL	7.4	86	36	11	5.140186916	0.7	133	4.8	134	N	N	N	N	1	0
9	44213	JAYAMMA	54	F	Y	N	N	Y	N	N	92	120	98	18	94	N	N	N	N	UL	7.36	89	46	28	13.08411215	0.5	125	3.7	124	N	N	N	N	2	0
10	85685	KRISHNAPPA	35	M	Y	N	Y	Y	N	N	99	112	98	28	89	N	N	N	N	ML	7.27	62	22	40	18.69158879	1.5	130	4.3	247	N	Y	N	Y	1	0
11	965758	DIWAKAR	45	M	Y	N	Y	Y	N	S,D	100	120	101	24	96	N	N	N	N	UL	7.4	87	29.5	18	8.411214953	0.8	139	4	88	N	N	N	N	1	1
12	981965	SARASWATHI	48	F	Y	N	N	Y	HTN,CCF	N	110	160	104	40	82	N	N	N	N	ML	7.38	110	60	10	4.672897196	0.4	117	4.8	160	N	N	N	N	2	1
13	974360	PUTTARAJU	34	M	Y	N	Y	Y	N	N	90	150	100	28	96	N	N	N	N	UL	7.4	88	56	15.8	7.38317757	0.3	134	4.1	112	N	N	N	N	1	1
14	975162	PARVATHIBAI	36	F	Y	N	N	Y	N	N	92	120	98	46	98	N	N	Y	N	UL	7.41	84	54	30	14.01869159	0.6	130	2.8	140	N	N	N	N	1	1
15	942320	KANAPPA	56	M	Y	Y	Y	Y	SPLENECTOMY	S	118	160	100	40	78	N	Y	N	Y	ML	7.34	87	43	29	13.55140187	14	138	3.5	94	N	Y	N	Y	3	1
16	40117	NARAYAN SWAMY	48	M	N	N	Y	Y	N	N	82	110	97	22	98	N	N	N	N	UL	7.58	103	18	34	15.88785047	0.6	138	3.5	545	N	N	N	N	1	1
17	117444	PADMA	28	F	Y	N	Y	Y	N	N	112	110	98	28	72	N	N	N	N	ML	7.24	69	50.6	58	27.10280374	0.8	136	3.6	120	N	Y	N	Y	3	1
18	45331	SRI RANGAPPA	85	M	Y	N	N	Y	HTN, DM,CCF	N	96	180	98	18	95	N	N	N	N	UL	7.41	85	42	42	19.62616822	2.1	135	4.6	180	N	N	N	N	3	2
19	44209	MANGAMMA	23	F	Y	N	N	Y	N	N	97	120	98	32	99	N	N	N	N	UL	7.41	78	34	26	12.14953271	1.6	138	4	110	N	N	N	N	1	1
20	44742	THIRUPATHAMMA	32	F	Y	N	N	Y	N	N	94	100	102	40	95	N	N	N	N	ML	7.36	89	39	45	21.02803738	1.4	129	3	124	N	N	N	N	2	1

# MASTER CHART

21	44795	ZAMRUTH BEE	66	F	Y	N	N	Y	DM	N	95	160	98	16	99	N	N	N	N	UL	7.4	86	32	41	19.1588785	1.2	134	4.5	384	N	N	N	N	3	2
22	115563	PANEER SELVAM	37	M	Y	N	Y	Y	N	N	120	160	100	35	60	N	N	N	Y	ML	7.47	34	32	42	19.62616822	1.6	141	5.1	86	N	Y	N	Y	3	1
23	100856	JAREEN TAJ	48	F	Y	N	Y	Y	HTN, DM	N	100	140	99.8	3	91	N	N	N	N	ML	7.4	90	60	100	46.72897196	3.1	128	5.8	164	N	N	N	N	2	2
24	976573	RAJANNA	60	M	Y	N	Y	Y	MI,CCF	S	110	140	98	60	72	N	N	N	N	ML	7.36	65	61	44	20.56074766	0.58	131	5	131	N	N	N	N	4	2
25	966416	VIMALAMMA	66	F	Y	N	Y	Y	HTN, DM	N	90	140	99	20	98	N	N	N	N	UL	7.37	89	43	35	16.35514019	1.5	135	4.1	112	N	N	N	N	2	3
26	979675	GOWRAMMA	29	F	Y	Y	Y	Y	N	N	140	110	100	48	100	N	N	N	Y	ML	7.1	59	60	24	11.21495327	1.8	131	4.7	90	N	N	N	Y	3	2
27	984561	MUNIYAPPA	63	M	Y	Y	Y	Y	N	N	100	150	102	40	89	N	N	N	N	ML	6.45	71	76	73	34.11214953	8.1	131	6	120	N	Y	N	Y	4	2
28	1009434	RATHNAMMA	60	F	N	N	Y	Y	HTN	N	140	160	99	30	89	N	N	N	N	ML	7.2	37	36	53	24.76635514	1.6	136	4.1	100	N	Y	N	Y	4	2
29	1016546	RAHTUNNUISA	52	F	N	N	Y	Y	HTN	N	110	100	99	35	94	N	N	N	Y	ML	7.26	79.4	61.8	34	15.88785047	1	136	4.3	120	N	N	N	N	1	2
30	108961	MANORMANI	47	F	Y	N	Y	Y	DM, HTN	N	128	180	100	30	80	N	N	N	N	ML	7.44	29	36.1	36	16.82242991	0.51	134	3.9	325	N	Y	N	Y	4	2
31	47671	KRISHNA SINGH	65	M	Y	N	N	Y	HTN,DM,CK D,CCF	S	85	160	99	24	86	N	N	N	N	ML	7.12	76	124	90	42.05607477	6.2	134	6.7	251	N	N	N	N	4	3
32	44706	YELLAMMA	75	F	Y	N	Y	Y	N	N	93	110	98	31	96	N	N	N	N	UL	7.42	78	42	36	16.82242991	1.5	112	4.7	80	N	N	N	N	3	3
33	112685	SARITHA	57	F	Y	N	Y	Y	DM, HTN	N	140	80	94	40	46	N	N	N	N	ML	7.4	29	36	36	16.82242991	0.5	136	3.6	270	N	N	Y	Y	3	2
34	112865	MANJAMMA	56	F	Y	Y	Y	Y	N	N	110	140	96	28	84	N	N	N	N	ML	7.46	57	24	12	5.607476636	0.7	124	3.8	117	N	N	N	Y	3	2
35	111905	MANOJ KUMAR	36	M	Y	N	Y	Y	N	S	120	110	98	44	74	N	N	N	N	ML	7.4	50	25	21	9.813084112	1.4	140	4.7	163	N	N	N	Y	1	2
36	100308	LAKSHMAMMA	60	F	Y	Y	Y	Y	DM	N	100	80	94	46	76	N	N	N	N	ML	7.2	48	80	90	42.05607477	3	128	4	286	N	Y	Y	Y	4	3
37	937031	RAMESH	30	M	Y	N	Y	Y	N	S	110	90	102	38	90	N	N	N	N	UL	7.1	56.5	58	113	52.80373832	14	127	5.4	112	N	Y	Y	N	4	3
38	952152	AMMAMMA	85	F	Y	Y	Y	Y	N	N	140	130	98	50	92	N	N	N	N	ML	7.32	58.4	36.8	28	13.08411215	0.47	132	3.8	156	N	Y	N	N	4	4
39	944182	NARAYAN SWAMY	60	M	Y	N	Y	Y	HTN	S,D	110	90	102	48	56	N	N	N	N	ML	7.4	78	55	80	37.38317757	3.2	127	3.8	94	N	N	Y	N	3	3
40	952130	THIPANNA	73	M	Y	Y	Y	Y	N	S,D	146	110	99	39	87	N	N	N	Y	ML	7.1	74	127	112	52.3364486	2.5	133	6.3	101	N	Y	N	Y	4	4
41	952132	THIPPAIAH	65	M	Y	N	Y	Y	HTN, DM	S,D	80	140	99	30	96	N	N	N	N	UL	7.4	100	56	45	21.02803738	0.9	143	3.3	180	N	N	N	N	2	4
42	100308	LAKSHMAMMA	60	F	Y	N	Y	Y	N	N	100	90	98	36	93	N	N	N	N	ML	7.35	86	27	101	47.19626168	10	141	4.5	103	N	N	N	N	3	3
43	6849	RAVI KV	57	M	Y	Y	Y	Y	HTN, DM	S,D	110	100	99	38	94	N	N	N	N	UL	7.1	75	152	154	71.96261682	3.6	131	6.8	180	N	Y	N	Y	4	3
44	98654	THIPPANNA	73	M	Y	N	Y	Y	N	S,D	146	110	99	39	87	N	Y	N	N	ML	7.1	65	127	112	52.3364486	1.2	133	6.3	101	Y	Y	N	N	5	4

# MASTER CHART

45	38734	GANGAMMA	65	F	Y	N	N	Y	HTN	N	86	140	98	32	99	N	N	N	N	UL	7.34	68	34	35	16.35514019	0.9	134	4.6	125	N	N	N	N	1	4
46	116532	MANJULA	35	F	Y	Y	Y	Y	N	N	120	120	98	44	66	N	N	N	N	ML	7.24	69	50	50	23.36448598	0.8	135	3.5	171	N	N	N	Y	3	3
47	939912	NAJMA BANU	22	F	Y	Y	Y	Y	N	N	145	60	98	48	74	N	N	N	N	UL	7.22	78	46.6	64	29.90654206	2.4	134	5.7	91	N	N	Y	Y	3	4
48	100680	THIMAKKA	35	F	Y	Y	Y	Y	N	N	120	80	102	35	87	N	N	N	N	ML	7.38	52	19	48	22.42990654	1.8	138	4.6	142	Y	Y	Y	Y	4	4
49	40568	GEETHA	47	F	Y	Y	Y	Y	DM, HTN	N	95	130	98	21	89	N	N	N	Y	ML	7.39	60	32.4	35	16.35514019	0.7	131	4.2	273	Y	Y	N	Y	5	4
50	123499	RAMAKRISHNAPPA	67	M	Y	N	Y	Y	N	N	109	80	102	24	74	N	N	N	N	ML	7.32	60	62.4	91	42.52336449	1.4	138	2.5	141	Y	Y	Y	Y	5	4
51	40661	GOVINDAPPA	65	M	Y	Y	Y	Y	HTN,CCF	S,D	110	80	96	36	68	N	N	N	Y	UL	6.85	147	68	161	75.23364486	3.2	142	4.2	200	Y	Y	Y	Y	5	5
52	164860	VENKATESH	50	M	Y	N	Y	Y	N	N	96	130	98	20	99	N	N	N	N	UL	7.35	186	40	38	17.75700935	1.5	142	3.5	124	N	N	N	N	1	0
53	170057	VEERAVATHI	24	F	Y	Y	Y	Y	N	N	160	50	93	42	20	N	N	N	N	ML	7.07	140	63.7	34	15.88785047	0.6	135	3.9	121	Y	Y	Y	Y	4	3
54	133837	VENKATAPPA	70	M	Y	N	Y	Y	N	N	84	90	96	30	98	N	N	N	N	ML	7.4	175	26	21	9.813084112	1.4	140	4.7	110	N	N	N	N	4	3
55	135977	BADRUNISA	55	F	Y	N	Y	N	DM	N	100	110	96	40	90	N	N	N	N	UL	6.74	164	75	79	36.91588785	2.3	133	4.4	252	N	Y	Y	Y	4	3
56	151199	CHIKKA Munireddy	80	M	Y	Y	Y	Y	DM, HTN	S	120	90	97	34	95	Y	N	N	Y	ML	7.35	127	30.6	98	45.79439252	2.6	139	4.6	114	Y	N	Y	Y	5	4
57	135964	NARAYANAPPA	65	M	Y	N	N	Y	N	N	92	110	100	20	96	N	N	N	N	UL	7.2	100	38	78	36.44859813	1.3	143	3.9	118	N	N	N	N	2	1
58	149095	SARDAR SAB	60	M	Y	N	Y	Y	N	N	135	98	93	40	95	N	N	N	N	UL	7.08	110	15.2	80	37.38317757	1.3	133	4	94	N	N	N	N	2	2
59	105315	NARAMMA	65	F	Y	N	Y	Y	N	N	98	90	99	28	86	N	N	N	Y	ML	7.12	69.2	28	23	10.74766355	1.1	124	4.4	156	N	N	Y	Y	2	2
60	133478	RAMAKKA	50	F	N	N	Y	Y	HTN	N	122	160	99	32	83	N	N	N	N	ML	7.4	94	40	59	27.57009346	1	116	7.4	216	N	N	N	N	3	2
61	33208	VENKATASWAMY	65	M	Y	N	N	Y	N	S	88	110	101	32	97	N	N	N	N	ML	7.42	97	69	217	101.4018692	9.3	134	5.8	130	N	N	Y	Y	3	3
62	33760	MANJUNATH	23	M	Y	Y	Y	Y	N	N	108	90	92	41	90	N	N	N	Y	ML	7.12	93	14.3	117	54.6728972	2.4	136	4.3	124	N	Y	Y	Y	3	3
63	128881	VENKATRAYAPPA	70	M	Y	N	N	Y	N	N	76	110	100	32	94	N	N	N	N	UL	7.42	78.5	39	36	16.82242991	0.9	142	3.6	81	N	N	N	N	2	1
64	92150	SOMASHEKHAR	40	M	Y	N	N	Y	N	S	125	120	99	26	93	N	N	N	N	UL	7.5	61	22.6	39	18.22429907	0.5	118	3.5	110	N	N	N	N	2	0
65	133260	RAMA BAI	65	F	N	N	N	Y	HTN, BA	N	90	160	99	18	98	N	N	N	N	ML	7.49	75.9	31.3	29	13.55140187	1.2	130	3.1	124	N	N	N	N	2	2
66	88144	PADMAMMA	32	F	Y	Y	Y	Y	N	N	120	50	95	60	85	N	N	N	Y	ML	7.2	92.9	40	58	27.10280374	1.6	116	4.1	146	N	N	Y	Y	3	3
67	85383	ANJANAMMA	45	F	Y	N	N	Y	N	N	96	80	98	42	80	N	N	N	N	ML	7.3	70.9	25.8	97	45.3271028	0.7	128	5.1	107	N	N	N	N	2	3
68	32012	YAHODAMMA	60	F	N	N	Y	Y	N	N	130	140	98	20	98	N	N	N	N	UL	7.41	60.2	34.1	66	30.8411215	1.1	138	4.3	124	N	N	N	N	2	1

# MASTER CHART

69	155629	MEENAKSHI	27	F	Y	N	Y	Y	N	N	80	110	100	16	98	N	N	N	N	UL	7.25	36.9	41.4	24	11.21495327	0.69	136	3.5	102	N	N	N	N	1	0
70	151543	MANJUNATH B	34	M	Y	Y	Y	Y	HTN, DM	N	115	170	102	34	30	N	N	N	Y	ML	7.54	119	30.6	16	7.476635514	0.74	138	5	451	N	N	N	Y	3	2
71	134320	CHINAPPA	70	M	Y	N	Y	Y	N	S	101	130	98.6	24	98	N	N	N	N	UL	7.3	104	40	28	13.08411215	0.74	141	4.3	91	N	N	N	N	2	1
72	118125	JAGANATH	34	M	Y	N	N	Y	N	D	82	120	99	28	85	N	N	N	N	UL	7.1	80	34	67	31.30841121	1.2	136	4.1	91	N	N	N	N	1	1
73	153367	PARVATHAMMA	56	F	Y	N	N	N	N	N	98	130	101	20	98	N	N	N	N	UL	7.3	104	42	40	18.69158879	0.92	134	3.5	124	N	N	N	N	2	0
74	156499	SHANKAR REDDY	55	M	Y	N	Y	Y	COR PULMONAL	S,D	104	80	97	34	74	N	N	N	N	UL	7.35	104	46	104	48.59813084	4.6	136	5.4	252	N	N	N	N	3	2
75	157467	RAMA REDDY	85	M	Y	Y	Y	Y	DM	N	140	70	96	32	75	N	N	N	N	ML	7.1	77	35	84	39.25233645	3.4	140	6	350	Y	Y	Y	Y	4	4
76	163725	SYED NAZIR	54	M	Y	Y	Y	Y	DM	S	50	90	97	28	70	N	Y	N	Y	ML	7.06	40.7	26.2	91	42.52336449	1.4	128	5.7	456	Y	Y	Y	Y	4	4
77	170804	NARENDRA NAYAK	24	M	Y	Y	Y	Y	N	N	144	60	94	50	20	N	N	N	N	ML	6.93	102	65	46	21.4953271	0.9	110	3.6	165	N	Y	Y	Y	3	3
78	173187	KENCHAPPA	75	M	Y	Y	Y	Y	DM,HTN,CK D,CCF	N	170	70	96	45	85	Y	N	Y	Y	ML	7.12	105	56	154	71.96261682	7.2	132	6.2	560	Y	Y	Y	Y	5	5
79	187598	VENKATESH	75	M	Y	Y	Y	Y	DM	D	132	80	102	46	75	N	N	N	N	ML	7.21	140	50	12	5.607476636	1.6	121	4.5	104	Y	Y	Y	Y	4	4
80	170552	KODANAPPA	40	M	Y	Y	Y	Y	N	N	128	70	97	60	50	N	N	N	N	ML	6.95	56	41	57	26.63551402	1.8	127	3.9	64	Y	Y	Y	Y	3	3