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Risk Stratification of Patients with Community Acquired Pneumonia Presenting to Emergency Room and Prediction of Mortality Based on Severity Scores

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Abstract: Community-acquired pneumonia (CAP) is defined as acute pulmonary infection in a patient, who is not hospitalized or residing in a long-term care facility for 14 or more Days before presentation. CAP is one of the most common infectious diseases addressed by clinicians and is an important cause of mortality and morbidity worldwide Pneumonia is one of the leading causes of death and morbidity, both in developing and developed countries and is the commonest cause (10%) of hospitalization in adult and children. Estimates of the incidence of community-acquired pneumonia range from 4 million to 5 million cases per year, with about 25% requiring hospitalization. Community-acquired pneumonia refers to pneumonia acquired outside of hospitals or extended-care facilities. It is important to risk stratify patients with pneumonia to look for morbidity and mortality. Objectives of study was to risk stratify the patients with community acquired pneumonia in the emergency room using CURB-65 score, SOFA score, QSOFA score, PSI score. All patients with community acquired pneumonia after application of the Inclusion and exclusion criteria were involved in the study. The clinical data with clinical Examination findings, investigations, clinical severity score, treatment, outcome were entered into a structured Performa. The patients were followed up for 28 days from the time of discharge. In our study we found that PSI and QSOFA score predicted mortality with p value of <0.001 which is highly sensitive compared to other scores.

Keywords: Pneumonia, CURB-65, Pneumonia Severity Index, Prognosis, Mortality, QSOFA

1. Introduction

Pneumonia is major cause of mortality and morbidity in both developing countries and developed countries.

Pneumonia is the major cause of death in children under five years and extremes of age [2]. Due to over usage and misuse of oral and intravenous antibiotics, patients are infected with multidrug resistant pathogens. Thiscan lead to healthcare associated pneumonia. Due to lack of knowledge, lack of facilities, pneumonia most often misdiagnosed, underestimated, and mistreated. One other reason for poor outcome of patients is failure to assess the severity of the disease and to treat patient as outpatients or in hospital setup or in intensive care unit. Pneumonia is more common in immunocompromised patients like Diabetes, HIV, and patients with chronic lung disease.

1.1. Epidemiology

The incidence of pneumonia in children younger than 5 years in India was 657 cases per 1000 children (95% UI 110–2357) in 2000 and 403 cases per 1000 children (74–1408) in 2015 [9]. The Estimated national pneumonia case fatality rate in 2015 was0·38% (95% UI 0·11–2·10) In spite of having a total number of deaths due to lower respiratory tract infections available, there is no systematic study conducted on the incidence of pneumonia in India [9]. According to the World Health Organization, in India Mortality due to infectious disease is caused by lower respiratory tract

infections is around 20%.

1.2. Pathogenesis

Lung Anatomy: Lung is a spongy structure which helps in purifying blood [2]. There are three lobes in the right lung and two lobes in the left lung. Left lung is smaller than the right lung. Lingual in the left lung is equivalent to the middle lobe of the right lung. Right Main bronchus is more vertical than the left one. Because of this reason, aspirated materials such as vomit, blood or any other foreign body mostly enters the right lung rather than the left lung. Both bronchi give rise to bronchioles.

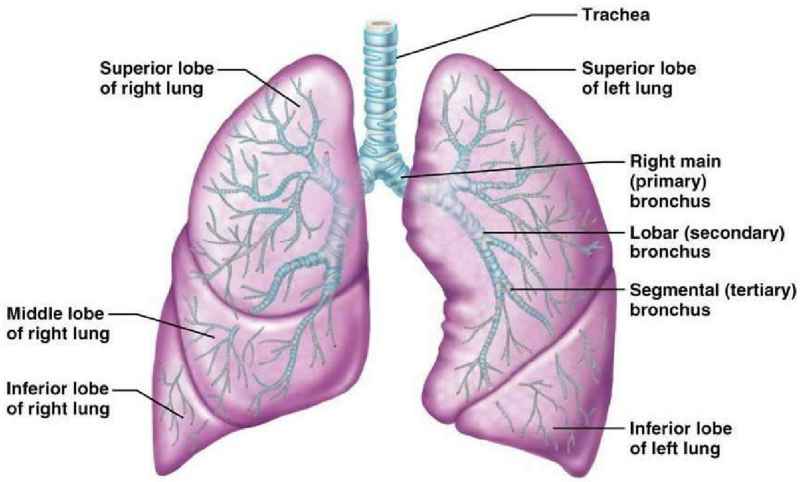

Figure 1. *Anatomy of Lung. Bronchioles are differentiated from bronchi by lack of sub mucosal glands and lack of cartilage. Bronchioles give rise to terminal bronchioles with diameter less than 2mm. distal to terminal bronchioles called as acinus, which are spherical approximately with 7mm diameter. Terminal bronchioles lead to respiratory bronchioles which proceed to alveolar ducts which branch to alveolar sacs.*

Alveolar sacs are blind ends where gas exchange takes place. Alveolar walls under microscope consist of, from blood to air,

The intertwining network of anatomising capillaries by the Capillary endothelium.

A basement membrane and surrounding interstitial tissue, which separates epithelial cells of alveolar lining from endothelial cells.

Alveolar epithelium, two cell types, continuous in nature.

95% of the surface is covered by type I pneumocytes, which are flattened and plate-like.

Type II pneumocytes [14], which are rounded. There are two major reasons why type IIpneumocytes are more important than type I pneumocytes14. They are i. After type I cell destruction, Type II cells are those, which helps in repair of destructed alveolar epithelium. ii. Type II pneumocytes are sources of pulmonary surfactant, in which an electron microscope contains osmiophilic lamellar bodies.

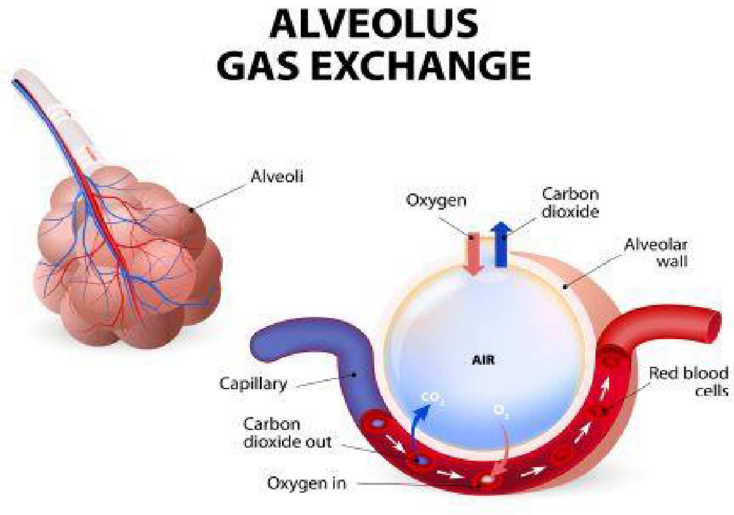

Figure 2. *Physiology of gas exchange in alveolus.*

Which may present free in alveolar spaces or attached loosely to epithelial cells. SomePhagocytosedmaterials and carbon particles are filled in alveolar macrophages. The walls of alveoli are not solid in nature but contain many Pores of

Kohn. Between adjacent alveoli, these pores allow bacteria and exudates [15]. Pneumonia is a common lung infection characterized by collection of pus and other fluids in the lung air sacs (alveoli). Lung air sacs are structures that help in the exchange of oxygen and carbon dioxide. Collection of pus in them makes breathing difficult. Pneumonia [5] can be caused by many kinds of microorganisms (germs) including bacteria, viruses, fungi, or parasites. When an infected individual coughs or sneezes, these organisms get into the air and breathing in of this air leads to contraction of the illness. It Is thus a contagious disease. It is of various types occurring in individuals of all ages, affecting millions of people worldwide.

The condition varies from mild to severe depending on the type of organism involved, age and the underlying health of the individual. Pneumonia can be categorized as: community acquired, hospital-acquired and pneumonia occurring in Immunocompromised individuals (individuals with weakened immune system).

1.3. Pathophysiology of Community Acquired Pneumonia

Pneumonia is an infectious process that occurs because of the invasion and overgrowth of microorganisms (as mentioned in the etiology part in this dissertation) in lung parenchyma, breaking down defense mechanisms [1]. It further provokes intra alveolar exudate production.

Basically, the development of pneumonia requires the pathogen to reach the alveoli and that host defenses are overwhelmed by microorganism virulence. The lungs are constantly exposed to particulate material and microbes that are present in the upper airway, from the air that is breathed in.

The lower respiratory tract can be entered by microorganisms by several mechanisms which include gross aspiration or micro aspiration of the oropharyngeal or gastric content, aerosolization of bacterial laden aerosol, hematogenous spread from a distant infected site and direct spread from a contiguous infected site. There are many determinant factors that can cause changes in the normal flora of the upper respiratory tract that predispose to infection, such as underlying disease, loss of mechanical respiratory defenses with the use of sedatives, tracheal intubation, and antibiotic treatments. In pneumonia, lungs capillaries become leaky, and protein-rich fluid seeps into the alveoli. This can lead to a less functional area for oxygen-carbon dioxide exchange, causing relative oxygen deprivation, while retaining potentially damaging carbon dioxide. The alveoli fill further with fluid and debris from the large number of white blood cells that are being produced to fight the infection.

Consolidation, a feature of bacterial pneumonias, occurs when the alveoli, which are normally hollow air spaces within the lung, instead become solid, due to fluid and debris. Pathogenesis of pneumonia involves: 1) congestion, which occurs in day 1 of infection due to vasodilation of the capillaries, 2) red hepatisation, which occurs in day 2, with accumulation of red blood cells and exudative production, 3) grey hepatisation, which occurs on day 4 of infection, with accumulation of neutrophils and macrophages, and 4) resolution, which occurs after day 8 with presence of few macrophages & normalization of lung parenchyma.

The pathology of pneumonia manifests as four general patterns which are lobar pneumonia, bronchopneumonia, interstitial pneumonia and miliary pneumonia. Lobar pneumonia classically involves an entire lung lobe relatively homogeneous, although in some patients, small portion of the lobe may be unaffected or at an earlier stage of involvement. Bronchopneumonia, a patchy consolidation involving one or several lobes, usually involves the dependent lower and the posterior portions of the lungs, a pattern that is attributable to the distribution of aspirated oropharyngeal content by gravity. Interstitial pneumonia predominantly involves the interstitium, including alveolar walls and the connective tissue around the bronchovascular tree.

Miliary pneumonia resembles the millet seeds in miliary tuberculosis due to hematogenous spread. Persistent and uncontrolled infection may lead to several complications such as abscess formation, necrotizing pneumonia, vascular invasion with infarction, cavitation, and extension to the pleura with effusion, empyema, or bronchopleural fistula.

Risk Factors for Community Acquired Pneumonia

There are a lot of factors which increase the risk of developing CAP including extremes of age, immunosuppressive diseases (e.g., diabetes mellitus, neoplasms, and HIV infection) respiratory disorders (e.g., bronchial asthma), use of drugs (e.g., oral steroids) and alcohol abusers. Age and comorbidities are known to be the risk factors for CAP [1].

Aging is associated with a decline in lung performance due to increase in elastic recoil of the lung, chest wall compliance and respiratory muscle strength. The mucociliary clearance, cough reflex and oropharyngeal deglutition are also impaired in the elderly and the ability to mount an immune response is abnormal due to impairment of T-cell function. While a study of severe CAP in 529 patients in 33 intensive care units inNot to forget, smoking is also one of the risk factors for getting pneumonia. Smoking alters the mucociliary transport, epithelial cell function and increases risk of adhesion of certain pathogens such as S. pneumoniae and H. influenza. Other than that, heavy alcohol use cause alterations of the immune system, impairs the function of lymphocytes, neutrophils, and other inflammatory cells, increasing host susceptibility to infectious disease, especially bacterialIncreasingly newer microbiological agents, some of which are well known, and some are very new pathogens, have revolutionized the understanding of pneumonia, and this led to the extensive use of modern antibiotics.

In the late twentieth and twenty-first century, newer microbial agents have emerged like - opportunistic lung infection in patients with HIV infection and post organ transplant patients. All these have led to an understanding of the immunological status of the individual. With the beginning of an antibiotic era, the mortality rate leveled off and remained constant. This mortality rate is heavily

weighted against elderly. [4]

2. Materials and Methods

Method of collection: All Patients with Community Acquired Pneumonia After Application of Inclusion And Exclusion Criteria Were Involved In The Study [16]. Study Duration Of One Year From July 7th 2022 ToAugust 7th 2023 In AJ Institute Of Medical Science Emergency Room Mangalore The clinical data with clinical examination findings, investigations, clinical severity score, treatment, outcome were entered into a structured proforma. The patients were followed up for 28 days from the time of discharge [7].

Collection of Data

(including sample size and sampling procedure) 65

Inclusion Criteria:

1) Age >18yrs
2) History of cough
3) History of fever
4) New focal sign on chest examination
5) Dyspnea
6) New radiographic shadow for which there is no other explanation [e g., not pulmonary edema, infarction]

Exclusion criteria:

Breathlessness due to other causes like acute respiratory distress syndrome, congenital heart disease, acute pulmonary embolism, pulmonary edema.

3. Study Design

A hospital based, prospective study

STUDY PERIOD:

1 year.

Statistical methods used:

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean±standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square ($\chi 2$) test was used for association between two categorical variables.

The sources of the variation include treatment; Error (a); the effect of Time; the interaction between time and treatment; and Error (b). Error (a) is the effect of subjects within treatments and Error (b) is the individual error in the model. All these add up to the total. If the p-value was < 0.05, then the results were statistically significant otherwise it was considered as not statistically significant. Data were analyzed using SPSSsoftware v.23 (IBM Statistics, Chicago, USA) and Microsoft office 2007.

4. Results

Table 1. *Distribution of cases according to age.*

AGE (YRS)	N	Percent
21-40	10	17.5
41-60	24	42.1
61-80	17	29.8
>80	6	10.5
Total	57	100

Table2. *Distribution of cases according to sex.*

SEX	N	Percent
Male	33	57.9
Female	24	42.1
Total	57	100

Table 3. *Distribution of cases according to presenting complaints.*

PRESENTING COMPLAINTS	N	Percent
FEVER	47	82.5
COUGH	48	84.2
DYSPNOEA	46	80.7

Table 4. *Distribution of airway.*

AIRWAY	N	Percent
PATENT	57	100
THREATENED	0	0
SECRETIONS	30	52.6

Table 5. *Distribution of b/l auscultation.*

B/L AUSCULTATION	N	Percent
CREPTS	51	89.5
DECREASED	6	10.5
Total	57	100

Table 6. *Descriptive statistics of breathing parameters.*

Descriptive Statistics	Min	Max	Mean	SD
RR	32	55	43.5	6.7
BMI	24	32	27.8	2.1
SPO2	53	89	69.6	10.1
HR	96	131	107.4	10.3
SBP	100	140	130.9	10.7
DBP	60	90	82.6	7.4

Table 7. *Descriptive statistics of disability and exposure Parameters.*

Descriptive Statistics	Min	Max	Mean	SD
GCS	13	15	14.7	0.6
GRBS	52	450	226.3	73.3
TEMPR	98	102	100.5	1.1

Table 8. *Descriptive statistics of blood parameters.*

Descriptive Statistics	Min	Max	Mean	SD
HB	5.6	15.7	12.0	2.3
RBC	2.3	5.1	4.1	0.8
WBC	1000	21620	9366.1	5199.4
PCV	16.8	47.1	36.2	6.8
PLT	0.98	4.49	2.3	1.0
NA+	114.8	147.2	129.2	8.3
K+	3.2	5.4	4.2	0.5
CL-	84.9	110.1	97.4	7.4
TBIL	0.3	7.5	1.3	1.7
DBIL	0.1	5.1	0.7	1.2
IBIL	0.1	2.4	0.6	0.6
U	17	94	48.3	20.7
C	0.5	3.29	1.1	0.6
PH	6.9	7.5	7.3	0.2
Pao2	28	89	70.8	15.2

Table 9. *Distribution of ecg.*

ECG	N	Percent
LVH	2	3.5
S. Rhythm	16	28.1
S. Tachy	39	68.4
Total	57	100.0

Table 10. *Distribution of allergies.*

ALLERGIES	N	Percent
No	51	89.5
Yes	6	10.5
Total	57	100

Table 11. *Distribution of rash.*

RASHES	N	Percent
No	50	87.7
Yes	7	12.3
Total	57	100.0

Table 12. *Distribution of past Illness.*

PASTILLNESS	N	Percent
HTN	35	61.4
IHD	33	57.9
T2DM	33	57.9

Table 13. *Distribution of HEAD TO TOE.*

HEADTO TOE	N	Percent
PALLOR	14	24.6
CYANOSIS	0	0
CLUBBING	3	5.3

Table 14. *Distribution of BMI.*

BMI	N	Percent
<25	4	7
25-30	49	86
>30	4	7
Total	57	100

Table 15. *Distribution of auscultation right.*

AUSCULTATION RIGHT	N	Percent
CREPTS	51	89.5
DECREASED	4	7
AE DECREASED	1	1.8
NVBS	1	1.8
Total	57	100

Table 16. *Distribution of auscultation left.*

AUSCULTATION LEFT	N	Percent
CREPTS	51	89.5
DECREASED	1	1.8
NVBS	5	8.8
Total	57	100

Table 17. *Distribution of chest X Ray.*

Chest X Ray	Right		Left	
	N	Percent	N	Percent
Hilar LN	0	0	0	0
Pleural Effusion	12	18	9	13.8
Consolidation	15	23	20	30.7
Cavity	5	7	8	12.3
ARDS/PulmEmbolism	10	15.3	10	15.3

Table 18. *Distribution of hospital mortality.*

In HospitalMortality	N	Percent
No	47	82.5
Yes	10	17.5
Total	57	100

Table 19. *Distribution of 28 days Mortality.*

28 Days Mortality	N	Percent
No	18	31.6
Yes	39	68.4
Total	57	100

Table 20. *Descriptive statistics of stays.*

DescriptiveStatistics	Min	Max	Mean	SD
No. of Hospital days	10	20	14.2	5.0
No. of ICU Days	4	14	9.3	4.2
No. of Ventilator Days	3	14	8.7	4.6

Table 21. *Distribution of score (Curb65).*

Score (Curb65)	N	Percent
1	12	21.1
2	34	59.6
3	11	19.3
Total	57	100

Table 22. *Distribution of Sofa.*

SOFA	N	Percent
4-5	37	64.9
6-7	18	31.6
≥8	2	3.5
Total	57	100

Table 23. *Distribution of score (Qsofa).*

Score (qSOFA)	N	Percent
1	27	47.4
2	30	52.6
Total	57	100

Table 24. *Distribution of PSI.*

PSI	N	Percent
50-100	28	49.1
100-150	24	42.1
>150	5	8.8
Total	57	100

Table 25. *Distribution of mean days according to score (curb65).*

Score (Curb65)	No. of Hospital days		No. of ICU Days		No. of Ventilator Days	
	Mean	SD	Mean	SD	Mean	SD
1	12.5	4.5	8.3	3.7	7.1	4.3
2	15.3	5.1	10.1	4.3	9.7	4.6
3	12.7	4.7	8.2	4.0	7.3	4.4

Score (Curb65)	No. of Hospital days		No. of ICU Days		No. of Ventilator Days	
	Mean	SD	Mean	SD	Mean	SD
p value	0.135		0.258		0.122	

Table 26. *Distribution of mean days according to Sofa.*

SOFA	No. of Hospital Days		No. of ICU Days		No. of Ventilator Days	
	Mean	SD	Mean	SD	Mean	SD
4-5	14.3	5.0	9.5	4.2	8.7	4.8
6-7	14.4	5.1	9.4	4.4	9.2	4.4
≥8	10.0	0.0	6.0	0.0	4.0	0.0
p value	0.483		0.527		0.324	

Table 27. *Distribution of mean Days according to score (Qsofa).*

Score (qSOFA)	No. of Hospital Days		No. of ICU Days		No. of Ventilator Days	
	Mean	SD	Mean	SD	Mean	SD
1	14.4	1.1	9.0	1.1	8.5	1.8
2	14.6	1.0	9.9	0.3	8.9	0.6
p value	0.040*		0.031*		0.039*	

Note: p value* significant at 5% level of significance (p<0.05)

Table 28. *Distribution of mean Days according to psi.*

PSI	No. of Hospital days		No. of ICU Days		No. of Ventilator Days	
	Mean	SD	Mean	SD	Mean	SD
50-100	10.0	0.0	5.8	1.5	4.8	1.0
100-150	17.9	4.1	12.5	3.1	12.2	3.6
>150	20.0	0.0	14.0	0.0	14.0	0.0
Total	14.2	5.0	9.3	4.2	8.7	4.6
p value	<0.001*		<0.001*		<0.001*	

Note: p value* significant at 5% level of significance (p<0.05)

Table 29. *In hospital mortality According to score (Curb 65).*

Score (Curb65)	In HospitalMortality			
	Yes		No	
	N	%	N	%
1	0	0.0%	12	25.5%
2	8	80.0%	26	55.3%
3	2	20.0%	9	19.1%
Total	10	100.0%	47	100.0%

Table 30. *In hospital mortality according to sofa.*

SOFA	In HospitalMortality				p value
	Yes		No		
	N	%	N	%	
4-5	6	60.0%	31	66.0%	0.470
6-7	3	30.0%	15	31.9%	
≥8	1	10.0%	1	2.1%	
Total	10	100.0%	47	100.0%	

Table 31. *In hospital mortality according to score (Qsofa).*

Score (qSOFA)	In HospitalMortality				p value
	Yes		No		
	N	%	N	%	
1	0	0.0%	27	57.4%	0.001*
2	10	100.0%	20	42.6%	
Total	10	100.0%	47	100.0%	

Note: p value* significant at 5% level of significance (p<0.05)

Table 32. *In hospital mortality according to psi.*

PSI	In HospitalMortality				p value
	Yes		No		
	N	%	N	%	
50-100	7	70.0%	21	44.7%	0.045*
100-150	1	10.0%	23	48.9%	
>150	2	20.0%	3	6.4%	
Total	10	100.0%	47	100.0%	

Note: p value* significant at 5% level of significance (p<0.05)

Table 33. *28 Days mortality according to Score (Curb 65).*

Score (Curb65)	28 Days Mortality				p value
	Yes		No		
	N	%	N	%	
1	7	17.9%	5	27.8%	0.475
2	23	59.0%	11	61.1%	
3	9	23.1%	2	11.1%	
Total	39	100.0%	18	100.0%	

Table 34. *28 days mortality according to Sofa.*

SOFA	28 Days Mortality				p value
	Yes		No		
	N	%	N	%	
4-5	24	61.5%	13	72.2%	0.533
6-7	14	35.9%	4	22.2%	
≥8	1	2.6%	1	5.6%	
Total	39	100.0%	18	100.0%	

Table 35. *28 days Mortality according to score (Qsofa).*

Score (qSOFA)	28 Days Mortality				p value
	Yes		No		
	N	%	N	%	
1	21	53.8%	6	33.3%	
2	18	46.2%	12	66.7%	0.149
Total	39	100.0%	18	100.0%	

Table 36. *28 days mortality according to psi.*

PSI	28 Days Mortality				p value
	Yes		No		
	N	%	N	%	
50-100	15	38.5%	13	72.2%	
100-150	21	53.8%	3	16.7%	0.029*
>150	3	7.7%	2	11.1%	
Total	39	100.0%	18	100.0%	

Note: p value* significant at 5% level of significance (p<0.05)

Table 37. *Roc Analysis Of Scores In Predicting In Hospital Mortality.*

Parameters	AUC	St. Error	p value	95%CI	
				Lower	Upper
Curb65	0.606	0.084	0.294	0.442	0.77
Sofa	0.651	0.076	0.136	0.503	0.799
qSOFA	0.787	0.061	0.005*	0.668	0.906
PSI	0.557	0.006	0.045*	0.549	0.766

Note: p value* significant at 5% level of significance (p<0.05)

Table 38. *Roc analysis of scores in predicting 28 days mortality.*

Parameters	AUC	St. Error	p value	95%CI	
				Lower	Upper
Curb65	0.587	0.08	0.295	0.429	0.745
Sofa	0.514	0.079	0.864	0.36	0.669
qSOFA	0.692	0.077	0.020*	0.541	0.844
PSI	0.729	0.072	0.006*	0.589	0.87

Note: p value* significant at 5% level of significance (p<0.05)

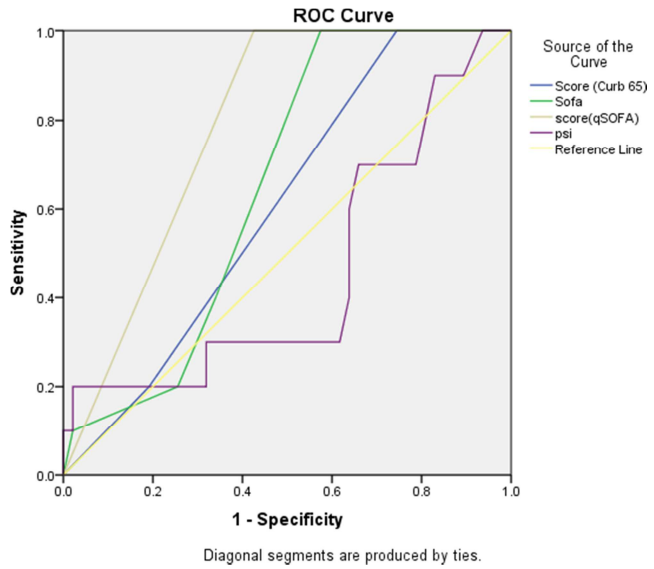

Figure 3. *Roc Analysis of Scores In Predicting in Hospital mortality.*

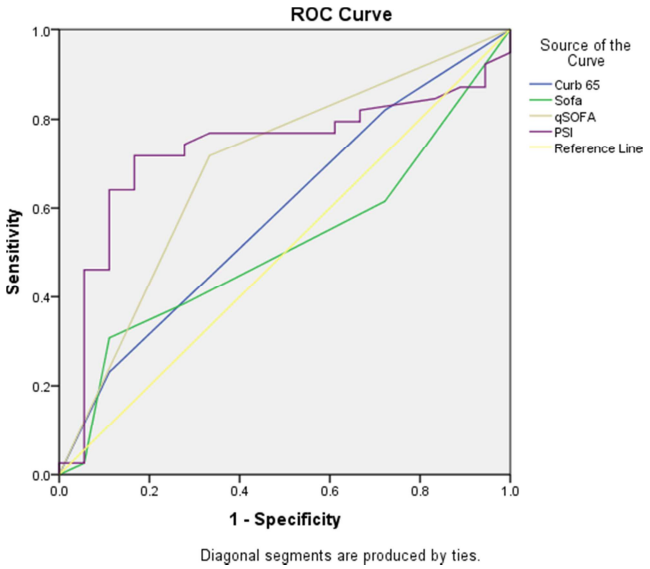

Figure 4. *Roc Analysis of Scores In Predicting 28 Days Mortality.*

5. Discussion

Despite being the cause of significant morbidity and mortality, pneumonia is often misdiagnosed, mistreated, and underestimated. Pneumonia is an infection of the pulmonary parenchyma and pneumonia that develops outside the hospital is considered as community acquired pneumonia [10].

Our study differed from other studies which showed a higher male preponderance among those admitted with CAP.CAP in elderly patients is associated [6] with high morbidity and mortality. The increased airway secretions could be attributed to the fact that the mucociliary clearance in increased in any respiratory infections. Measuring the respiratory rate is an important and simple tool for assessing the severity of acute cardio respiratory and metabolic diseases.

One of the most basic and easiest predictive methods of worsening CAP is the estimation of the respiratory rate. Those patients who are found to be tachypneic objectively measured by a RR>= 30/min can reflect a severe disease process.

Admission blood pressure (BP) assessment is a central component of severity assessment for community [6] acquired pneumonia30 Admission systolic BP < 90 mmHg, diastolic BP < or = 60 mm Hg, mean arterial pressure < 70 mm Hg and pulse pressure < or = 40 mm Hg were all associated with increased 30 day mortality and the need for mechanical ventilation and/or inotropic support. Similar observations were made in our study with mean systolic blood pressure of 130mm Hg and mean diastolic BP of 82.6. We also found that the mean heart rate of patients in our study was 107 which indicates tachycardia is a common sign in any respiratory illness or CAP.

An acute state of confusion is one of the most well studied parameters used in the stratification of CAP. An acutely confused patient is more likely to present late in the

disease progression and have associated sepsis. The total numbers of patients who present with confusion in most studies are low however there is a definite association of confusion as an adverse predictor of outcome and 66 severity of the disease. More than half of our study population had diabetes mellitus and the Mean grbs was 226.3 at the time of presentation.

The involvement of the lung parenchyma in CAP represents a compromise to the respiratory function of the target organ [11]. A low PaO2/FiO2 in patients with CAP is one of the pathophysiologic signs of lung impairment and poor outcome. In our study we found that mean serum urea was 48.3 and. mean serum creatinine was 1.1 the use of creatinine as a surrogate marker to determine pneumonia needs further validation similar to Urea >7 mmol/L which has been validated by a study by Lim et al in 2000 [17].

Urea is easily reabsorbed in the kidney hence it tends to overestimate the glomerular filtration rate (GFR) especially in cases with dehydration. Serum creatinine is now frequently used to assess GFR in AKI.

According to the RIFLE and AKIN classifications of AKI a two three fold increase in Creatinine is needed to indicate injury to the kidney.

Detailed examination of respiratory system is very crucial in any CAP. even though it is difficult to get all the detailed examination points in ER, we were able to find few important respiratory examination findings. In our study on examination of respiratory system 89.5% of patients had bilateral crepitations on auscultation /In our study 8.8% of patients had normal vesicular breath sounds on auscultation. Followed by decreased air entry in 1.8 % of patients.

In our study we have categorized chest x ray finding in right and left lung.18% of patients had pleural effusion on right side and 13% on left side. We also found that 53% of our study population had consolidation in either of the lung fields. cavitary lesions were found in 19% of the population. ARDS was present in 30% of patients at the time of presentation to ER.

In our study, we discovered that the average number of days spent in the hospital was 14.2, with a standard deviation of 5.0. and we also found that Mean no of ICU days was 9.3 with sd of 4.2 and Mean no of ventilator days was 8.7 with SD of 4.6. A variety of factors were linked to prolonged hospitalization in patients with community-acquired pneumonia: the most common were pneumonia-related clinical and nonclinical factors, followed by complications and underlying condition. Initial hypoxemia, anemia, neoplastic illness, and complications occurring within 72 hours of admission were all clinical variables linked to a longer stay. In ur study we analyzed the QSOFA score of all the study population during their presentation to ER.

Based on these observations we would like to conclude that QSOFA score has significant value in assessment and prediction of number of ICU days and ventilator days for any CAP patients. even though QSOFA score has its own drawbacks in application and selection of patients, if it is properly used in isolated CAP patients in ER, it has significant role in predicting the hospitalization and in hospital mortality. However QSOFA score does not aid in predicting the 28 day mortality. The Pneumonia Severity Index (PSI) [3] is a useful scoring system for determining the severity of community-acquired pneumonia and the need for hospitalization. The PSI class denotes mortality, with a higher score indicating a greater CAP mortality rate.

The PSI risk factors individually associated with increased risk of death included lower systolic blood pressure; increased respiratory rate; altered mental status; and the presence of acidosis, lower hematocrit, elevated blood urea nitrogen, hyponatremia, and a history of either congestive heart failure or cerebrovascular disease. The PSI covariates that were associated with increased risk of death. Other desirable characteristics of the PSI are that it directly yields a patient-specific estimate of mortality risk, uses information readily available for most patients, and uses predictors measured at or near the time of admission.

In our research, we discovered that the P value for the number of hospital days, ICU days, and ventilator days was 0.001, indicating that it is highly sensitive in predicting morbidity and death.

The p value for 28-day mortality according to the psi score was 0.029, whereas the p value for in-hospital mortality according to the psi score was 0.045.

The CURB-65 can be used in the emergency department setting to risk stratify a patient's community acquired pneumonia. CURB 65 is calculated, and each feature is assigned one point (range 0–4 points). 42 CURB-65 is simple to calculate, requiring information about the patient that is almost certainly already available, and gives excellent risk stratification for community-acquired pneumonia. It has the potential to improve resource use and treatment start. The CURB-65 [13] score has the advantage of providing a wider range of specificity sensitivities, allowing patients to be classified as potentially eligible for three alternative therapeutic approaches. Patients with scores of 0 and 1 have a low risk of death (2%), and may be managed as outpatients in the hospital or by their primary care physician. Patients With a score of 2 have an intermediate risk of mortality (9%) and should be referred to a hospital for treatment.

This establishes that each of the components of the CURB score is a predictor of The CURB score was categorized as <2 or >2 or =2 in this model, a score of >2 or =2 is being taken to identify patients with severe CAP.

6. Conclusion

CAP is a prevalent cause of sepsis and a typical presentation to the emergency department (ED) with a high fatality rate. [8]

The goal of our study is to look into the prognostic value of SOFA, QSOFA, PSI, and CURB65 in septic patients with CAP who present to the ED and to predict mortality based on these scores, to see which score predicted well after 28 days. We also looked into the morbidity status of patients in terms of hospital, ICU, and ventilator days during their hospital

stay.

The combined predictive value of sequential organ failure assessment (SOFA) and rapid SOFA (QSOFA), [8] PSI, and CURB 65 for CAP had not been researched before, and if studied separately, only a maximum of three scores had been compared. Our study is one of the few that has looked at the predictive performance of CURB65, CRB65, PSI, SOFA, and QSOFA all at once. The QSOFA and PSI scores were found to be superior in predicting 28-day death, ICU admission, mechanical ventilation, and morbidity.

These tools remain accurate for predicting mortality, but are not direct measures of disease severity and cannot supersede clinical assessment to determine the need for hospital admission or ICU care. These tools have their greatest value for allowing comparison between different CAP clinical studies, so that the population studied can be better characterized, and the outcomes can be compared, in relation to predicted mort.

A combination of QSOFA and SOFA scores was found to be the strongest predictor of pneumonia severity and outcome in a few studies. and only a few with both PSI andCURB65. The CURB-65 is simple, and useful for preventing clinicians from overlooking vital sign abnormalities that define severe illness. However, it is not ideal for detecting patients with multiple co morbid illnesses, especially if these illnesses are decompensated by the presence of CAP.

We applied SOFA scores for isolated CAP patients presenting to ER, at the time of presentation, however the results were not statistically significant in prognostication of the CAP patients. [12]

CURB65 for mortality prediction, while its discriminative power decreased with advancing age. The PSI was developed to predict low-risk patients, but it is complex to use, and may not be as valuable for identifying critically ill individuals. It may overestimate mortality risk in old patients with comorbidity, while underestimating need for ICU care in younger patients who have not been previously ill. The QSOFA is determined by three vital signs: respiratory rate, systolic pressure, and altered consciousness. In our study we analyzed the QSOFA score as a strong predictor of the study population during their presentation to ER. Based on these observations we would like to conclude that QSOFA score has significant value in assessment and prediction of number of ICU days and ventilator days for any CAP patients. The P value for QSOFA and PSI in predicting the number of hospital days, icu days, and ventilator days were significant, indicating that it is highly sensitive in predicting morbidity and death, according to our findings.

Thus we conclude that PSI and QSOFA SCORE were highly helpful in predicting mortality in our study compared to other scores.

7. Objectives of the Study

Risk stratification of patients with community acquired pneumonia in the emergency department using CURB-65 SCORE, SOFA, QSOFA, PSI. Evaluation of the above clinical severity scores for community acquired pneumonia to predict mortality at 28 days. Materials and Methods: Source of data: Patients who present to the emergency department of AJ institute of medical science and research center during the study period. Method of collection: All patients with community acquired pneumonia after application ofInclusion and exclusion criteria were involved in the study. The clinical data with clinical Examination findings, investigations, clinical severity score, treatment, outcome were entered into a structured Performa. The patients were followed up for 28 days from the time of dischargeCollection of data: (including sample size and sampling procedure) 65. Results: We have studied patients based on their presentation to ER with four scores, QSOFA, SOFA, PSI and Q SOFA. Interpretation and Conclusion: In our study we found that PSI and QSOFA score predicted mortality with p value of <0.001 which is highly sensitive compared to other scores.

Conflicts of Interest

The authors declare no conflict of interest.

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