"STUDY OF ACUTE ILLNESS OBSERVATION SCALE AND SERUM PROCALCITONIN LEVELS IN COMMUNITY-ACQUIRED PNEUMONIA IN CHILDREN AGED 2 MONTHS TO 59 MONTHS"

BY

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UNDER THE GUIDANCE OF

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ABSTRACT

Introduction

Community-acquired pneumonia (CAP) poses a major health challenge worldwide, with notable variations in incidence, causative pathogens, and outcomes across different regions and populations. In India, CAP accounts for about 23% of the global burden, with an estimated 4 million cases annually, 20% requiring hospitalization. The annual incidence ranges from 5 to 11/ 1,000 individuals, with varying mortality rates based on the setting:

Neuroendocrine cells found in the thyroid, lung, and intestine typically produce procalcitonin (PCT), a protein precursor to the hormone calcitonin. As part of the innate immune system's pro-inflammatory response, parenchymal cells release PCT, a precursor peptide of calcitonin, which is detectable 4 hours after endotoxin stimulation—much sooner than CRP. Procalcitonin (PCT) has been established as a marker of bacterial infection to differentiate septic from other infections in pediatric patients. PCT is less than 0.5 ng/ml in healthy people and rises quickly within three hours of the bacterial infection developing. Within six to twelve hours, PCT levels reach their peak and stay elevated until the infection subsides due to either antibiotic therapy or the host's immunological response.

To the best of our knowledge, there has not been a comparable study conducted on a pediatric population in India, which is why this study was selected.

Objectives

1. To evaluate the child according to the parameters of the Acute Illness Observation Scale (AIOS) and score the condition of the child.
2. To measure the Serum Procalcitonin levels and correlate the values with the AIOS score.

Materials And Methods

It's a Prospective Cohort Study conducted in Pediatric ICU, Shri. B. M. Patil Medical College, Hospital & Research Centre, BLDE (DU), Vijayapura, Karnataka with a duration of 1.5 years with a sample size of 51 and Children between the ages of two and fifty-nine months who exhibit fever, cough, and hurried breathing were included in the study.

Statistical Analysis

Data collected was entered into a Microsoft Excel spreadsheet and analyzed using **SPSS software.** The results were expressed as Mean (Median) ± Standard Deviation (SD), Interquartile range (IQR), Counts and percentages. **Graphical Representation:** Findings were illustrated using appropriate diagrams and charts.

Results

Out of 51 children, the majority were in the age group of 12 months -5 years i.e., 52.9% (n-27) followed by 47.1% (n-24) in the age group of <12 months, 64.7% (n-33) were a male child and 35.3% (n-18) were a female child. We found that 56.9% (n-29) had moderate AIOS on day 1 and then 62.7% (n-32) had Mild AIOS on day 2 followed by 72.5% (n-37) had mild on AIOS on day 5. We found that there is a statistical association between AIOS score and procalcitonin level and also with Procalcitonin and CRP level (p-value <0.05).

Conclusion

Both the Acute Illness Observation Scale (AIOS) along Serum Procalcitonin levels demonstrate a strong ability to detect the severity level and determine clinical outcomes in children with community-acquired pneumonia. Hence, we conclude that combining the AIOS scoring system along with Serum Procalcitonin levels has a better clinical decision. The mortality risk increases when patients have high AIOS scores and their procalcitonin levels remain elevated.

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LIST OF ABBREVIATIONS

ABBREVIATIONS	EXPANSION
AIOS	Acute Illness Observation Scale
CCP 1	Calcitonin Carboxyl-terminus Peptide I
CALC1	Calcitonin I
CAP	Community-Acquired Pneumonia
HAP	Hospital-Acquired Pneumonia
PCT	Procalcitonin
RSV	Respiratory Syncytial Virus
RISC	Respiratory Index of Severity in Children
TNF	Tumor Necrosis Factor
UNICEF	United Nations Children's Fund

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Introduction

Community-acquired pneumonia (CAP) poses a major health challenge worldwide, with notable variations in incidence, causative pathogens, and outcomes across different regions and populations.

Globally, CAP leads to 1.4 million visit the emergency department, 740,000 hospitalizations, and 41,000 deaths annually. The incidence is higher among older adults (≥65 years) and individuals with underlying health conditions. [1]

In India, CAP accounts for about 23% of the global burden, with an estimated 4 million cases annually, 20% requiring hospitalization. The annual incidence ranges from 5 to 11/ 1,000 individuals, with varying mortality rates based on the setting. [1,2]

Causative Pathogens

The etiology of CAP varies geographically and is influenced by local epidemiology and healthcare practices. Globally, *Streptococcus pneumoniae* is a predominant pathogen. In India, studies have identified *S. pneumoniae, Klebsiella pneumoniae*, and *Staphylococcus aureus* as common causative agents. Notably, the rising incidence of *K. pneumoniae* in India warrants attention due to its association with severe disease and antibiotic resistance. [1]

A significant contributor to morbidity in both the developed and developing worlds is pediatric respiratory illness. [2] Knowledge Acquired A public health concern, pneumonia is the main cause of death for children between the ages of two and fifty-nine months. It is now the most frequent reason why parents contact a pediatrician and the most frequent cause why patients are admitted to hospitals.

Community-acquired Pneumonia is a respiratory infection caused by bacteria, viruses, or other pathogens. It affects children and is caused by a wide range of organisms and is an infection of the lung parenchyma. The elderly and young children are particularly vulnerable, but anybody can be impacted. Most of the time, a cold or flu spell of illness precedes and causes pneumonia. [2,3]

Optimizing triage, early referral, hospitalization, and treatment criteria is crucial because India has one of the highest rates of pneumonia-related mortality. [4,5] The technique known as Integrated Management of Neonatal and Childhood Illness (IMNCI) has made this easier by streamlining the process of classifying the severity of key acute childhood illnesses, such as pneumonia.

When combined with an illness severity scoring can rapidly measure the severity of disease at every step from onset to recovery, the IMNCI approach will be more successful. P.L. McCarthy created the generic illness severity scale known as the Acute Illness Observation Scale (AIOS) in 1982, which is based on straightforward observations rather than intricate symptomatology. It has been proven to be helpful in this regard. [6,7] The six easily observable components of AIOS are a sensitive indicator of serious disease in children when combined. AIOS's overall score falls between 6-30. [8] If a child has a fever and scores 10 or lower, the likelihood of a serious bacterial infection is less than 2-3%; if scores are between 11 and 15, it is 26%; and if the AIOS score is 16 or higher, it is 92%. [8]

Because proponents assert that biomarkers may enhance the early detection of infections and be accessible as a point-of-care tool, their usage in clinical practice has significantly expanded [9]. Patients with serious, perhaps fatal infections may be able to be identified and treated sooner and more effectively as a result. Neuroendocrine cells found in the thyroid, lung, and intestine typically produce procalcitonin (PCT), a protein precursor to the hormone calcitonin. [10, 11] As part of the innate immune system's pro-inflammatory response, parenchymal cells release PCT,

a precursor peptide of calcitonin, which is detectable 4 hours after endotoxin stimulation—much sooner than CRP. Procalcitonin (PCT) is recognized as a biomarker for bacterial infections, aiding in the distinction between bacteria and other types of infections in children. The molecular weight of the peptide procalcitonin (PCT) is approximately 13 kDa, and it has 116 amino acid residues. In 1993, PCT was presented as a new and creative infection metric. The standard endocrine theory states that the main source of PCT is the neuroendocrine C cells in the thyroid. Its production is regulated by the calcitonin I (CALC-I) gene on chromosome 11p15.2-p15.1. In healthy individuals, plasma PCT was found to be very low. [10]

Higher PCT concentration has been linked to the isolation of common bacterial species in numerous adult trials, particularly when it comes to bacterial CAP. [11,12] However, children do not have the same evidence. While other research indicated a somewhat strong correlation between PCT content and organism identification in CAP, two investigations in children reported a limited correlation. The sensitivity of PCT tests, organism identification, and testing procedures hampered a lot of earlier research. [13]

Studies have shown that PCT is increased in bacterial rather than viral infections. Hence, it can be used for judicial use of antibiotics in children and reduce resistance to antibiotics.

PCT is less than 0.5 ng/ml in healthy people and rises quickly within three hours of the bacterial infection developing. Within six to twelve hours, PCT levels reach their peak and stay elevated until the infection subsides due to either antibiotic therapy or the host's immunological response.

To the best of our knowledge, there has not been a comparable study conducted on a pediatric population in India, which is why this study was selected.

AIMS & OBJECTIVES:

1. To evaluate the child according to the parameters of the Acute Illness Observation Scale and score the condition of the child.
2. To measure the Serum Procalcitonin levels and correlate the values with the AIOS score.

REVIEW OF LITERATURE:

Pneumonia is a frequent lung illness with potentially dangerous consequences. It can be brought on by a number of microorganisms, such as bacteria, viruses, fungi, and even some chemicals, and it causes swelling and inflammation of the air sacs in one or both lungs. Bacteria or viruses are the most common causes of pneumonia, an acute respiratory disease. Despite being the world's largest infectious cause of mortality for children, it can infect people of all ages and cause mild to fatal illnesses. [14]

Acute respiratory infection: [14]

Any upper or lower respiratory illness as described by the International Classification of Diseases falls under this category. Acute lower respiratory infections, which include serious infections like pneumonia, impact the airways beneath the epiglottis. When it comes to acute lower respiratory infections, pneumonia is responsible for a considerable amount of the disease burden.

This report uses the term "pneumonia" to refer to "suspected pneumonia." In situations with limited resources, definitive confirmation by laboratory testing or radiography is typically absent, therefore a suspected case of pneumonia is detected by its clinical symptoms.

Therefore, a cough and rapid or labored breathing are synonymous with probable pneumonia in all children under five. The two further classifications for suspected pneumonia patients are "severe" and "non-severe."

CAP, or community-acquired pneumonia: [24] According to clinical definitions, CAP occurs

when a previously healthy child develops pneumonia-like symptoms as a result of an infection contracted outside of the hospital. This is confirmed by the radiological discovery of consolidation in affluent nations. Because getting an X-ray can be challenging in developing nations, the more practical phrase acute lower respiratory tract infection is recommended.

EPIDEMIOLOGY

More than 700,000 children under the 5 years of age die from pneumonia each year, or around 2,000 every day, making it the infectious disease that kills most children. Approximately 190,000 infants are included in this. Nearly all of these fatalities might have been avoided. Over 1,400 instances of pneumonia per 100,000 children, or one case per 71 children, occur worldwide each year; South Asia and West and Central Africa have the highest incidences, with 2,500 and 1,620 cases per 100,000 children, respectively. [(15)] Compared to other infectious diseases, the rate of decrease in pneumonia-related fatalities in children under five has been much slower. Diarrhoea deaths have decreased by 63% since 2000, and they now account for nearly half of pneumonia deaths in children under five. In contrast, pneumonia deaths have fallen by 54% since 2000.

Only five nations—China, Nigeria, India, Pakistan, and the Democratic Republic of the Congo—account for around half of all deaths among children under five. Together, Nigeria (13 percent) and India (22 percent) are responsible for over one-third of all deaths in children under five. [(15)] According to recent estimates by the United Nations Children's Fund (UNICEF), pneumonia remains the leading cause of death for children worldwide, accounting for 18% of all child fatalities and an estimated 1.3 million child deaths in 2011 alone. 8 In the first two years of life, 81% of deaths linked to pneumonia and 72% of deaths linked to diarrhea occur.

(15)

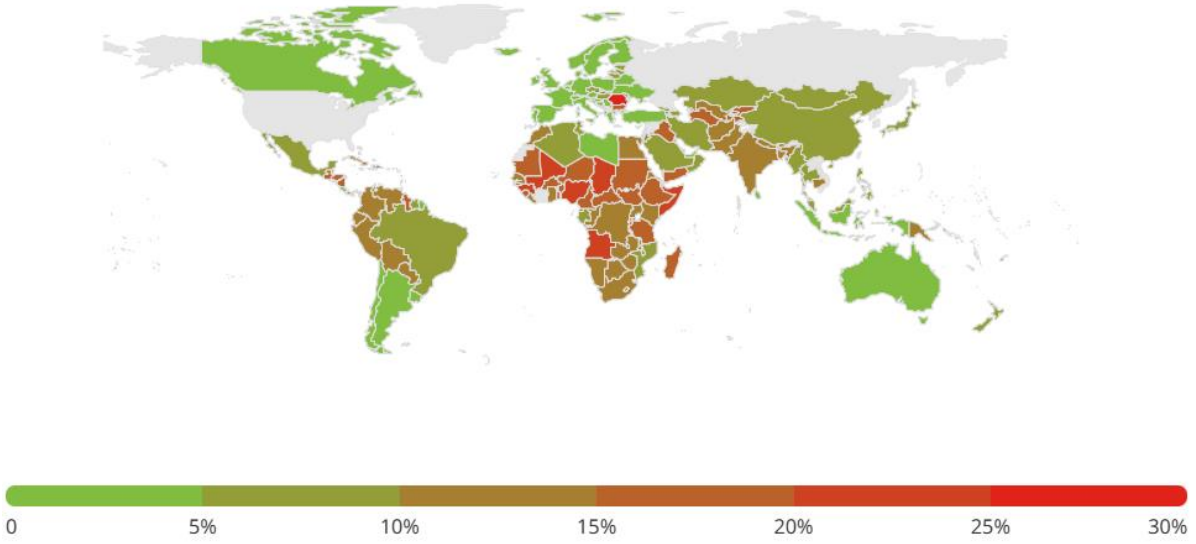

FIGURE 1: Global Under-five mortality rates

Malnutrition, a lack of access to safe drinking water and sanitation, indoor and outdoor air pollution, and poverty are all factors that contribute to the mortality rate of childhood pneumonia. By 2030, an additional 18 million health workers are expected to be needed to prevent, diagnose, and treat pneumonia as well as to meet the Sustainable Development Goals for universal health care. Air pollution is responsible for approximately half of all pediatric pneumonia deaths; indoor air pollution kills more children globally than outdoor air pollution, and more than 2 billion children between the ages of 0 and 17 reside in areas with outdoor air pollution levels that are higher than recommended levels.

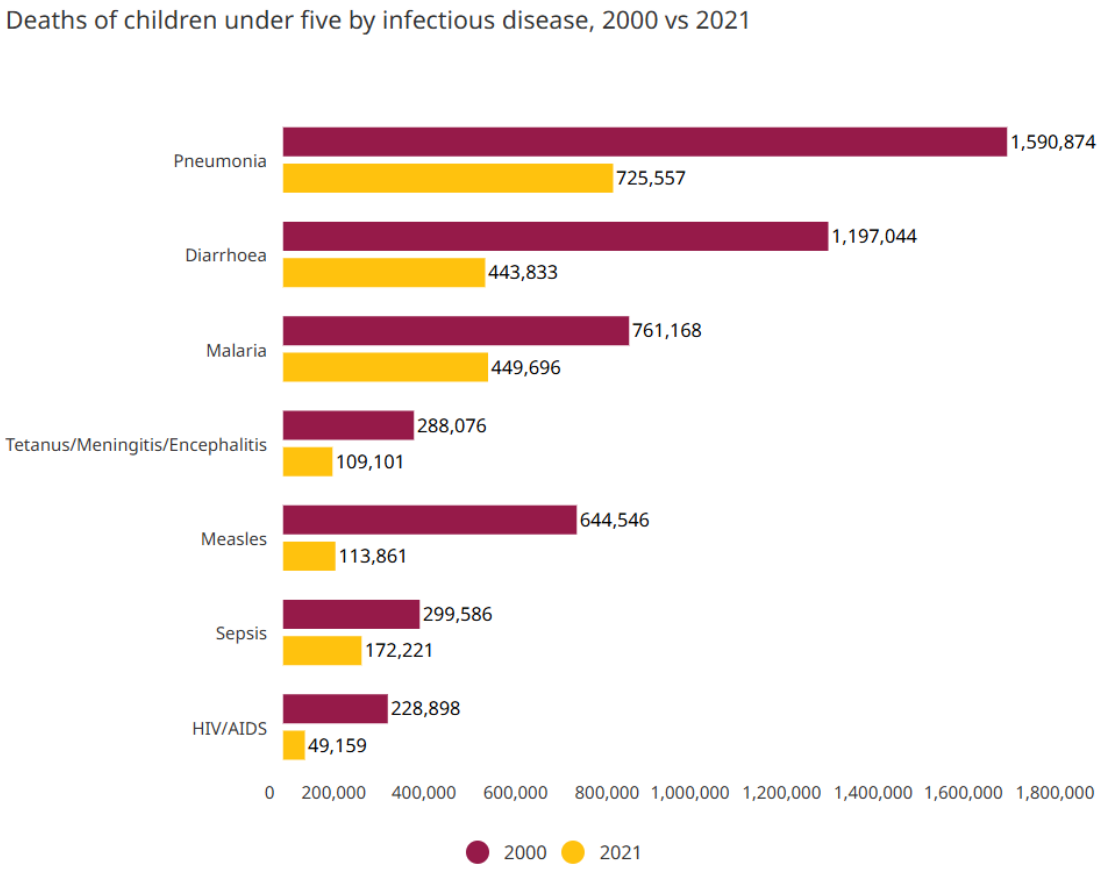

FIGURE 2: Causes of death in under five population in the world

Although CAP-related morbidity and mortality can occur in any region, 95% of these cases occur in underdeveloped nations (Figure 2). With only 15 nations accounting for 74% (115.3 million occurrences), it remains the leading cause of death for children in low-income countries. India now accounts for 43 million of the world's yearly new CAP cases, followed by China with 21 million, Pakistan with 10 million, Bangladesh, Indonesia, and Nigeria with 6 million apiece. [16]

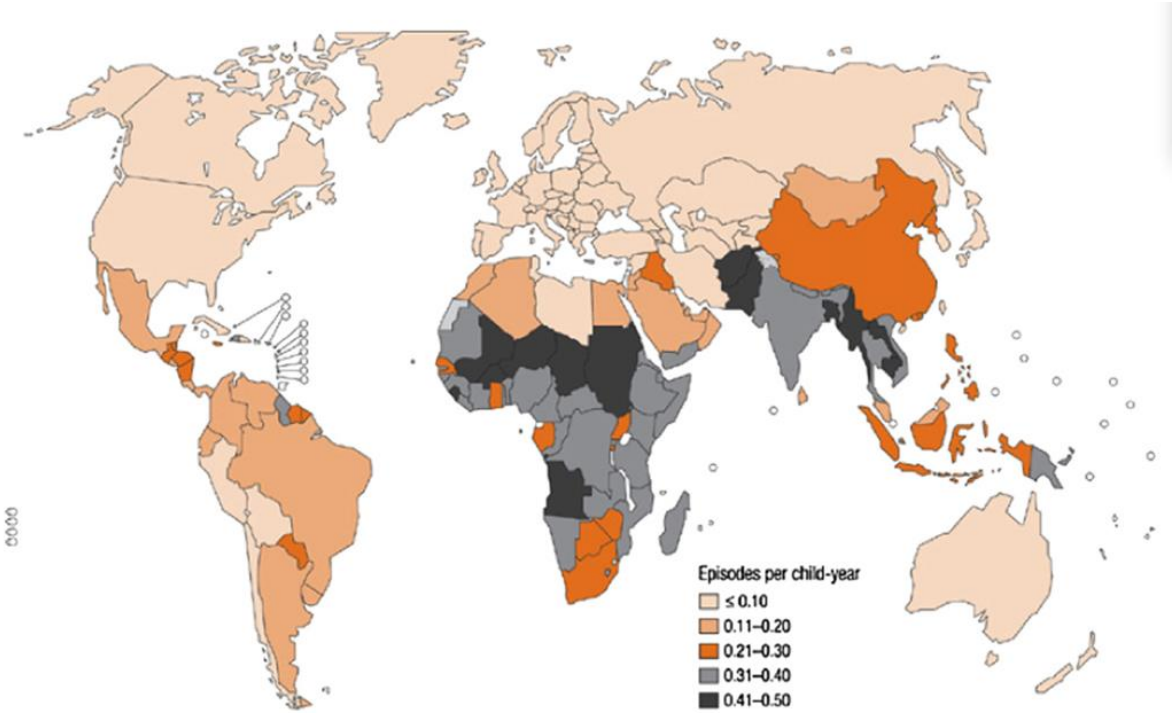

FIGURE 3: Incidence of childhood clinical pneumonia at the country level

PATHOPHYSIOLOGY OF COMMUNITY-ACQUIRED PNEUMONIA [17]

Viral Pneumonia

Viral pneumonias occur when the viral pathogen or pathogens directly infect the upper respiratory tract through inhalation of infectious secretions or from environmental fomites. The virus spreads distally to the lower respiratory tract, where it multiplies, de-epithelializes, and inhibits ciliary activity, causing mucus to block the airways and debris to accumulate. Following the virus's progress to the alveoli, cells degrade, resulting in the loss of surfactant production, the development of hyaline membranes, and perhaps pulmonary edema. Oedema is exacerbated and gas exchange across alveolar membranes is reduced when mononuclear cells invade the submucosal and interstitial regions. [18,19]

Through a ball-valve mechanism, mucus and cellular debris obstructing the bronchioles can

cause air trapping and hyperinflation, especially in cases of RSV pneumonia. This mechanism can also lead to hypoxemia, ventilation-perfusion mismatch, and segmental or subsegmental atelectasis. Viral pneumonia in children can be caused by a number of circumstances and can vary in severity. Children who have underlying lung conditions, such as chronic lung disease or bronchopulmonary dysplasia, which cause them to have trouble clearing their elevated secretion volume, may experience atelectasis, hypoxemic respiratory failure, and increasing bronchospasm and wheezing. Because of their narrow airways and lack of Kohn pores, which connect alveolar spaces, infants are more vulnerable to more severe viral pneumonia. Wheezing and atelectasis can also result from these disorders. Last but not least, deficiencies in the humoral or innate immunity that are essential for protecting against viral infections may make viral pneumonia more severe.

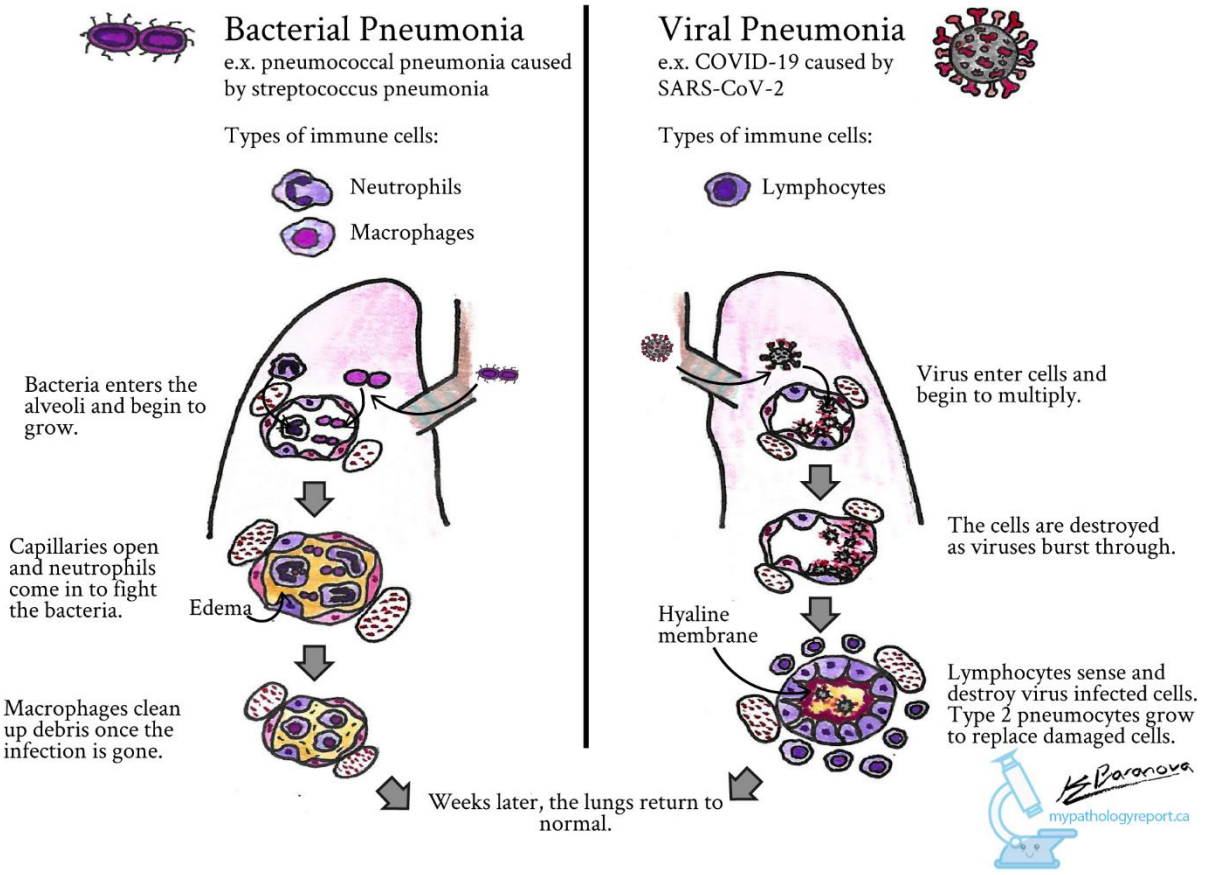

FIGURE 4: Pathogen of CAP

Bacterial Pneumonia

The lung's defences against bacterial pathogens include the reticuloendothelial system, which stops hematogenous spread, alveolar macrophages, which engulf and kill airway bacteria, ciliated epithelium and mucus, which trap and remove organisms from the airways, and innate and specific humoral immunity, which defuses infecting bacteria. Bacteria may enter the lungs, multiply, and start destroying cells; if any of these barriers are broken, which can lead to pneumonia. Most cases of bacterial pneumonia follow colonization of the nasopharynx. [20] With de-epithelialization raising the possibility of bacterial colonization, this could happen after a viral upper respiratory infection.

The most frequent bacterial cause of pneumonia in children is *Streptococcus pneumoniae*. Once colonized, bacteria can have hematogenous spread, aspirate, or inhale their way into the lower respiratory system. Pneumococci attach themselves to the bronchioles, proliferate, and emit exudative fluid, which starts an alveolar inflammatory cascade, known as "red hepatization,". Fibrin is deposited and polymorphonuclear cells infiltrate the alveoli, which is followed by an increase in macrophage activity, also known as "white hepatization." Exudative fluid contributes to the typical pattern of lobar pneumonia by enabling the germs to grow and disseminate to nearby alveoli. More tissue is destroyed and pulmonary abscesses and necrosis are more frequent when pneumonia is brought on by more virulent organisms, including S. aureus.

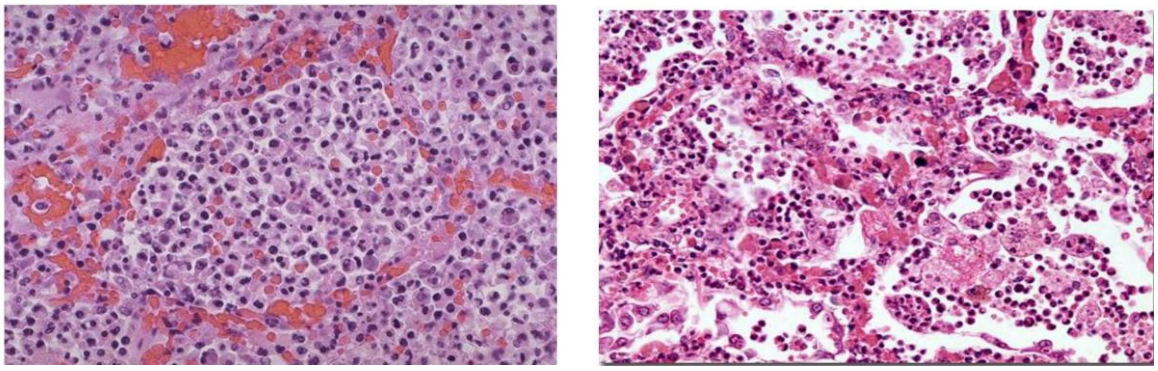

FIGURE 5: Schematic diagram showing red and grey hepatization

Atypical Pneumonia [23]

Aerosolized particles carry the mucosal pathogen *Mycoplasma pneumoniae*. When the bacteria attach, a cytokine response occurs, drawing neutrophils and lymphocytes to the mucosal cells and causing inflammatory infiltration of the airways. Oxidative stress follows the attachment of Mycoplasma to the respiratory epithelium, resulting in the epithelium's loss of function and occasionally necrosis. Diffuse alveolar injury or bronchiolar or alveolar edema may be part of the lung damage pattern.

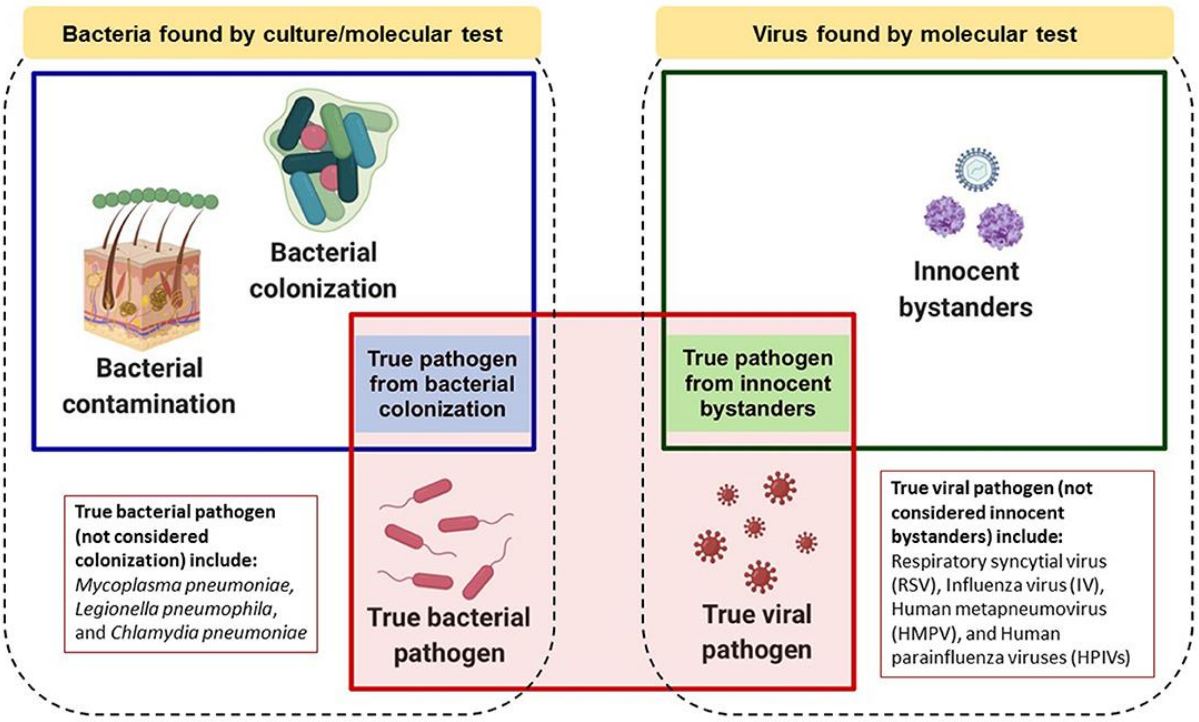

FIGURE 6: Etiology of Community-Acquired Pneumonia (CAP)

PATHOPHYSIOLOGY OF COMMUNITY-ACQUIRED PNEUMONIA (CAP) IN CHILDREN [24]

Community-acquired pneumonia (CAP) in children occurs when a pathogen from the external environment infects the lower respiratory tract, leading to inflammation and consolidation of lung tissue. The pathophysiological process of CAP in children is similar to adults in terms of immune response but differs due to the unique characteristics of the paediatric immune system, respiratory anatomy, and the pathogens involved.

1. Initial Pathogen Invasion

- Exposure to Pathogens: In children, CAP is typically caused by bacteria, viruses, or, less commonly, fungi. The most common pathogens include:
    - Bacteria: *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Staphylococcus aureus*, and *Mycoplasma pneumoniae*.

- Viruses: Respiratory Syncytial Virus (RSV), influenza, parainfluenza, and adenovirus.

- Entry through Upper Respiratory Tract: Pathogens usually enter the respiratory system through the nose or mouth and travel into the lower respiratory tract, especially in younger children who have less efficient airway clearance.

- Immature Immune Response: The immune system in young children is not fully mature, which can make them more susceptible to infections. They may not have the fully developed mucociliary clearance, phagocytic function, or the ability to generate specific immune responses as efficiently as adults.

2. Infection and Inflammatory Response

Once the pathogen reaches the alveoli, the immune system responds:

- Activation of Immune Cells: The alveolar macrophages recognize the pathogen and activate the innate immune response, including the release of pro-inflammatory cytokines (e.g., interleukins, tumour necrosis factor). This leads to inflammation of the lung parenchyma.

- Recruitment of White Blood Cells: Neutrophils and other immune cells are recruited to the site of infection. These cells attempt to clear the pathogen by phagocytosis and also release enzymes and inflammatory mediators, which contribute to tissue damage and further inflammation.

- Exudative Phase: The inflammatory response leads to increased vascular permeability, causing fluid to leak from blood vessels into the alveoli. This fluid, known as exudate, consists of white blood cells, proteins, and cellular debris, which impair normal gas exchange.

Community-acquired pneumonia has four stages: [25]

The initial phase, which lasts for 24 hours, is distinguished by vascular congestion and alveolar edema. There are neutrophils and bacteria.

The second stage, known as red hepatization, is similar in consistency to the liver. Red blood cells, neutrophils, and desquamated epithelial cells are characteristics of the stage. Alveolar fibrin deposits are frequent.

Two to three days later, the lung looks dark brown in the third stage of grey hepatization. Red cell hemolysis and hemosiderin buildup are present. The cellular infiltrates are resorbed and the pulmonary architecture is restored during the fourth stage, which is known as the resolution stage. Parapneumonic effusions may result from suboptimal healing.

Causative Organisms: [26]

Pneumonia is a lung infection that can be caused by a variety of microorganisms. These include bacteria, viruses, fungi, mycoplasma, and other agents, each of which may result in different clinical presentations. Below is an overview of the most common causes of pneumonia:

Pneumonia is most commonly caused by *Streptococcus pneumoniae (pneumococcus)*, although other bacteria such as Haemophilus influenzae,

Pneumonia can also result from Legionella pneumoniae and Staphylococcus aureus.

Viruses: Three types of viral agents that can cause viral pneumonia include influenza viruses, respiratory syncytial viruses (RSV), and coronaviruses.

Fungi: Usually affecting those with compromised immune systems, fungal pneumonia is a quite uncommon illness. Examples include Aspergillus species and Pneumocystis jirovecii, which are prevalent in HIV/AIDS patients.

Atypical pneumonia is the term for Mycoplasma pneumonia, which presents with relatively distinct symptoms and physical indicators. The bacterium Mycoplasma pneumoniae is the cause of it. All age groups are typically affected by the mild, widespread pneumonia it creates.

Other Agents: Additionally, aspirating (inhaling) food, liquids, or chemicals can occasionally result in pneumonia.

Based on a number of factors, such as the causative agent, the site of infection, and the environment in which it was contracted, pneumonia can be divided into many categories.

The primary forms of pneumonia are as follows: [26]

1. **Community-Acquired Pneumonia (CAP):** One of the most prevalent types of pneumonia, CAP is contracted outside of medical facilities. Numerous bacteria, viruses, and even fungi can cause it. The most frequent bacterial cause of CAP is Streptococcus pneumoniae. Influenza and respiratory syncytial virus (RSV) are examples of viral causes.
2. **Nosocomial pneumonia, or Hospital-Acquired Pneumonia (HAP),** is a condition that develops 48 hours or more after a patient is admitted to the hospital. It usually affects those who are already sick or have debilitated, and it is frequently more severe than CAP.
3. **Viral pneumonia**

   Lung infection brought on by a virus. Although the viral flu is the most frequent cause, other viruses and the common cold can also cause viral pneumonia. These harmful bacteria typically adhere to the upper respiratory tract. However, when they enter your lungs, pneumonia sets in.

4. **Fungal pneumonia**

   A non-contagious lung disease called fungal pneumonia can be brought on by fungal spores. It happens when the spores mix with the air and are inhaled, or when an inert infection is reactivated. Fungal pneumonia can cause flu-like symptoms such as coughing, headaches, thick mucus, fever, and chest pain. Fungal pneumonia is an extremely uncommon illness that typically affects people with weakened immune systems, such as those with HIV/AIDS, organ transplant recipients, or chemotherapy patients. Pneumocystis jirovecii pneumonia (PCP) is a well-known form of fungal pneumonia that commonly affects people with compromised immune systems.

5. **Opportunistic pneumonia**

   Incidental Bacteria known as opportunistic pathogens frequently do not infect healthy individuals with strong immune systems. One type of lung infection known as opportunistic pneumonia is caused by pathogens.

6. **Bronchopneumonia:**

   This form of pneumonia is characterized by inflammation of several tiny lung tissue regions surrounding the bronchi and bronchioles (small airways). An uneven spread of inflammation is frequently the outcome. The distinction between pneumonia kinds may not always be obvious, and it is crucial to keep in mind that classifications might occasionally overlap. Treatment and diagnosis rely on determining the source of pneumonia, the patient's health, and other pertinent variables. Also referred to as localized pneumonia/ lobular pneumonia.

7. **Pneumonia from Aspiration**

   A lung infection known as aspiration pneumonia is brought on by breathing in food, drink, vomit, saliva, or even tiny foreign objects. With the right drugs, it can be cured. Complications can be severe, even lethal, if untreated. Aspiration pneumonia is

pneumonia brought on by an inhalation of something other than air into your respiratory system. A tiny foreign object, food, drink, saliva, stomach contents, or poisons can all be considered non-air substances.

Clinical decisions regarding the necessity of hospitalization, intensive care unit (ICU) admission, and treatment planning are heavily influenced by an accurate assessment of the severity of the condition.

Numerous scoring schemes have been created and approved for this use over time. The usefulness of the Acute Illness Observation Scale (AIOS) in evaluating CAP is covered in this paper, along with an outline of regularly used scales.

1. **CURB 65** [27]

   First presented by Lim et al. (2003), the CURB-65 score is a popular and easy-to-use method for determining the severity of CAP. It assesses five factors: age ≥65 years, blood pressure, respiration rate, urea levels, and disorientation.

   According to a study by Pedro Carlos, 96.9% of the study group consisted of high-risk individuals (CURB>2). Similar to earlier research, the inpatient mortality rate of 13% increased to 21.5% after six months and was associated with the entrance CURB-65 score. Streptococcus pneumoniae isolates were found in 53% of cases, and a microbiologic agent was found in 37% of cases. Numerous more studies have continuously shown how well it predicts mortality and directs treatment choices. It is especially prized for being user-friendly in environments with minimal resources, as it requires minimal laboratory data.

2. **The PSI, or Pneumonia Severity Index [28]**

   A more thorough method that divides patients into five risk classes according to demographics, comorbidities, clinical findings, and test data is the PSI, which was created by Fine et al. (1997)

   According to a study by Mi-AeKim, whether or not there was a respiratory viral infection, there was an increase in mortality rate as there was an increase in PSI score. Mortality corrected for respiratory virus detection was substantially correlated with the pulmonary severity score (hazard ratio = 1.024, 95% CI = 1.020–1.028). Regardless of the presence of a respiratory virus, the pneumonia severity index score is a crucial component in determining the prognosis of patients with community-acquired pneumonia.

3. **SMART-COP [28]**

   Charles et al. (2008) developed the SMART-COP score to predict whether CAP patients would require intensive respiratory or vasopressor assistance. SMART-COP provides a dynamic method for identifying high-risk individuals by combining data including arterial pH, albumin levels, and systolic blood pressure. Research has demonstrated that it is more sensitive than PSI and CURB-65 at predicting intensive care unit needs.

4. **The severity of the respiratory index (RISC) [29]**

   Ekta Verma's study, which was conducted in a hospital, sought to assess how well the respiratory index of severity in children (RISC) score predicted the severity and mortality risk of pediatric pneumonia patients in a poor country. The RISC score is a potentially helpful tool for forecasting the severity and mortality risk of juvenile

pneumonia patients, according to the study.

5. The AIOS, or Acute Illness Observation Scale [32]

The AIOS is a behavioral observation tool that was first created by P.L. McCarthy [32] in 1982 for use with young populations. It uses behavioral and physical signals to determine the severity of the disease. Its usage in adult respiratory infections has attracted interest recently, despite not being traditionally utilized for CAP. The significance of non-laboratory-based clinical markers, like alertness, respiratory effort, and overall look, is emphasized by AIOS. According to studies examining its applicability in CAP, AIOS may be able to supplement other scales in situations where laboratory testing is either delayed or not accessible. To prove its effectiveness in adult CAP populations, further thorough validation research is necessary.

In comparison to pulse oximetry alone, an AIOS score of more than 10 is more sensitive and less specific in predicting abnormal X-rays, according to a study by Mudiganti Abhitej Reddy. [31] A poor clinical course, complications, a longer hospital stay, and culture-positive pneumonia are all substantially correlated with an AIOS score of greater than 15. The treating physician can utilize AIOS scoring to predict the length of hospital stay that may be necessary for a kid who has been admitted with pneumonia, in addition to using it to choose therapeutic modalities.

According to a study by Vinod Kumar Mishra, the AIOS score can be used to predict abnormal X-ray findings in children aged 2 to 59 months who have respiratory illnesses. [32]

According to a study by Anoop K, AIOS scoring has excellent external validity, internal consistency, and interobserver agreement between two observers. Peripheral healthcare professionals can utilize IMNCI as a tool for early referral and triage of children with community-acquired pneumonia in the field. A doctor may utilize AIOS to predict the prognosis and choose treatment options for a hospitalized child with pneumonia. [4]

Scale used	Acute illness observation scale
Items included	Quality of cry
	Response to parent stimulation
	State variation
	Colour
	Hydration
	Response to social overtures
Score interpretation	Each item scored as normal (=1)
	Moderate (=3) and severe
	Impairment (=5)
Total score	6= best score
	30= worst physical score

Observation item	1(Normal)	3(Moderate impairment)	5(Severe impairment)
Quality of cry	Strong with normal tone or content and not crying	Whimpering or sobbing	Weak or moaning or high pitched
Reaction to parent stimulation	Cries briefly then stops or content and not crying	Cries off and on	Continual cry or hardly responds
State variation	If awake, stays awake or if asleep and stimulated, wakes up quickly	Eyes close briefly awake or awakes with prolonged stimulation	Falls to sleep or will not rouse
Color	Pink	Pale extremities or acrocyanosis	Pale or cyanotic or mottled or ashen
Hydration	Skin and eyes normal and mucous membrane moist	Skin and eyes normal and mouth slightly dry	Skin doughy or tented & dry mucous membrane & or sunken eyes
Response (talk, smile) to social overtures	Smiles or alert(≤2mo)	Brief smile or alerts briefly(≤2mo)	No smile or face anxious, dull, expressionless or no alerting (≤2mo)

A study on the relationship between AIOS scores, chest X-ray results, and pulse oximetry readings in community-acquired pneumonia was carried out in Wayanad, India, by Pavan D et al. According to a descriptive epidemiological survey, the majority of patients (57.2%) are between the ages of 2 and 12 months. 54% show abnormal chest X-ray results, such as infiltrates (59.7%) and consolidation (42.2%). Of the 67 children who had SpO2 levels above 92% upon admission, 44 (65.67%) had normal chest X-ray results, while 23 (34.32%) had abnormal results. Lower SpO2 levels are linked to higher AIOS scores. [33]

Sikha Maria Siromani et al. conducted a study in Hyderabad, India on Prospective study on AIOS score and its correlation with radiological findings and WHO grading of ARI in admitted

patients. The relationship between AIOS and WHO grading was linearly positive and the association between the two had a significant P value. The relationship between AIOS score and the length of stay was also significant. Compared to children that were discharged, children who died had higher mean AIOS score at the time of admission. Hence, the authors concluded that AIOS score is helpful to predict the severity of the disease and predict abnormal Xray findings in children. [34]

PROCALCITONIN

Procalcitonin (PCT) has emerged as a promising novel biomarker for the early diagnosis of systemic bacterial infections in clinical practice today. Le Moullec et al. initially described PCT, a 116-amino acid residue, in 1984; nevertheless, it wasn't until 1993 that its diagnostic value was acknowledged. [34] Assicot et al. showed in 1993 that patients with positive results for sepsis and bacterial infection (e.g., positive blood cultures) were positively correlated with high serum levels of PCT. Additionally, they showed that blood levels of PCT would drop after the administration of suitable antibiotic treatments and that PCT would not increase in viral infections. [35,36] Antimicrobial therapy should not be determined solely by PCT. [37, 38] A PCT assay's results should be interpreted within the clinical context, taking into account the severity of the disease, the possibility of bacterial infection, the potential site of infection, and other relevant clinical information.

Biochemistry and Physiology of PCT

The 116-amino acid peptide PCT, which has a molecular weight of 14.5 kDa, is produced by the calcitonin 1 gene (CALC-1) on chromosome 11. It is divided into three parts: calcitonin

carboxyl-terminus peptide 1 (CCP-1), also known as katacalcin (21 amino acids), immature calcitonin (33 amino acids), and the amino terminus (57 amino acids). Pre-PCT, the gene's product, undergoes proteolytic cleavage to produce PCT, which is then converted into the mature calcitonin molecule [39]. Thyroid C-cells and, to a lesser extent, other neuroendocrine cells are often the only cells that can translate and transcribe the CALC-1 gene. However, in response to bacterial infection, production is triggered in all parenchymal tissues through the cytokines interleukin-6 (IL 6), tumour necrosis factor-α (TNF-α), and interleukin-1β (IL β). [39] PCT builds up in these other tissues because they are unable to split it into its mature form, calcitonin. [40] In contrast, interferon-γ, which is mostly released in reaction to viral infection, attenuates PCT synthesis. Because of this feature, PCT is a more precise indicator of bacterial infection. [41]

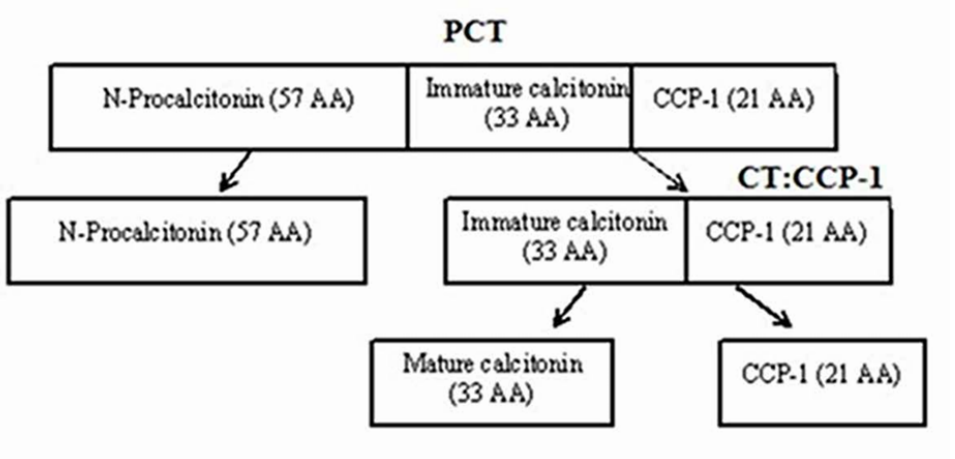

FIGURE 7: Biochemistry and Physiology of PCT

PATHOPHYSIOLOGY

Thyroid C cells are responsible for the early synthesis of pre-procalcitonin under normal homeostasis. Later, endopeptidases cleave a 25-amino acid signal sequence in this peptide,

converting it to procalcitonin. After being converted by the enzyme prohormone convertase, the 32-amino acid hormone that controls serum calcium, calcitonin, is the final result. [42] Serum PCT levels under healthy conditions are typically very low, less than 0.05 ng/ml. The circulating endotoxins or cytokines like interleukin (IL)-6, tumor necrosis factor (TNF)-alpha, and IL 1b, can boost the production of PCT by 100–1000 times. [43] The liver, pancreas, kidney, lung, gut, and leukocytes are the sites of extra-thyroid PCT synthesis; interestingly, the production of PCT is inhibited in these tissues when there is no bacterial infection. [44] On the other hand, PCT will be down-regulated by cytokines like interferon (INF)-gamma that is generated during viral infection, underscoring yet another benefit of PCT tests. [45]

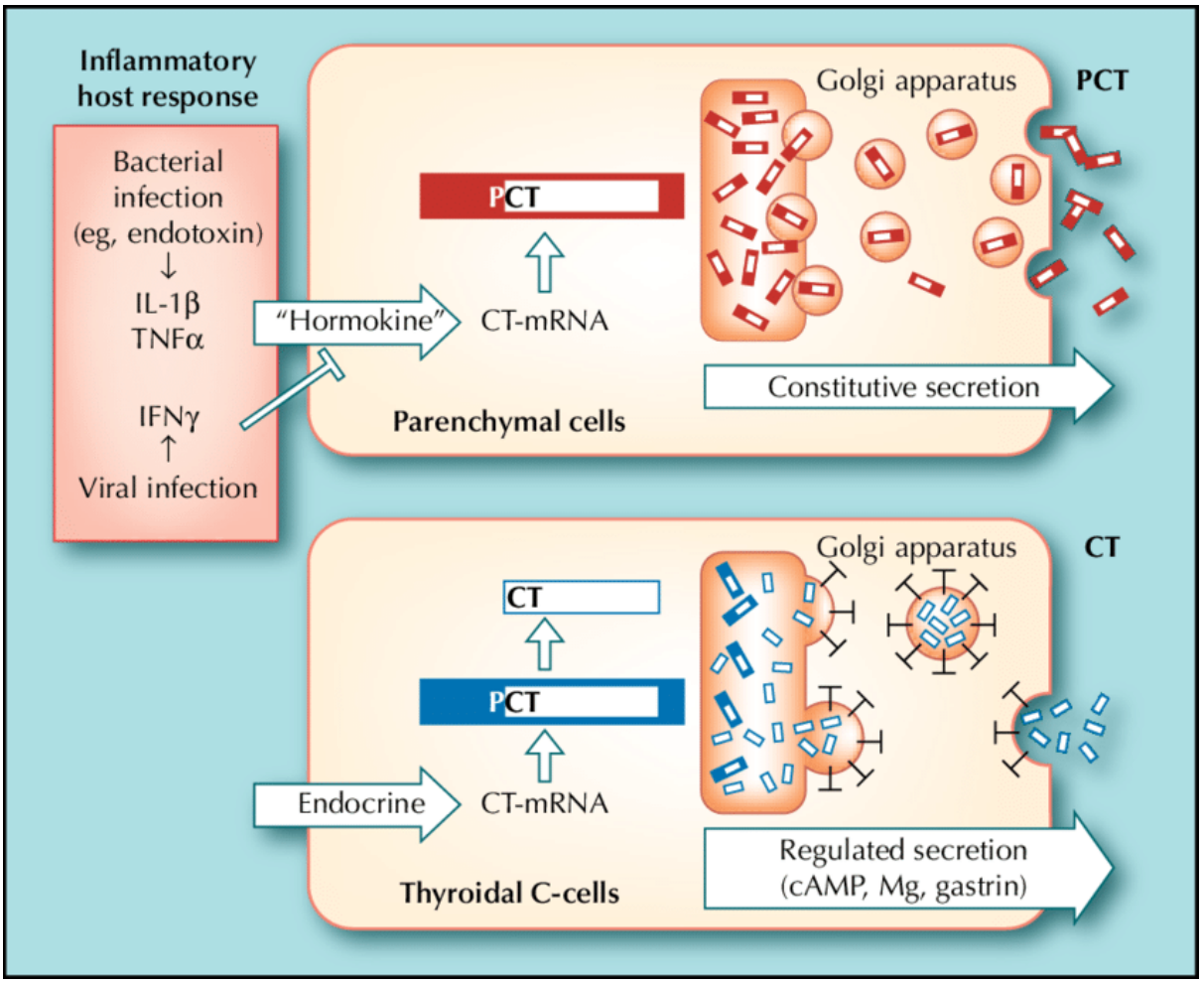

FIGURE 8: Interpretation of PCT values

Interpretation Of PCT Values

In healthy people, the serum PCT content is normally less than 0.1 μg/L. PCT rises when a bacterial infection is present, and the degree of the rise is correlated with the infection's severity. Compared to patients with septic shock, severe sepsis, and generalized sepsis, those with localized infections show lower increases in PCT. A decreasing concentration typically indicates that the illness has resolved. [46, 47]

PCT (μg/L)	Interpretation
< 0.05	Healthy adult
0.05 – <0.5	Systemic infection is unlikely although localised infection is possible
0.5 – <2	Systemic infection is possible but other conditions (e.g. major trauma, recent surgery, severe cardiogenic shock) may also induce significant PCT rises.
2 – <10	Systemic infection is likely
≥ 10	High likelihood of severe bacterial sepsis or septic shock

Role of procalcitonin on Community-acquired Pneumonia

Procalcitonin (PCT) is a biomarker that has been extensively studied for its role in diagnosing and managing community-acquired pneumonia (CAP) in children. Its utility spans diagnosis, assessing disease severity, and guiding antibiotic therapy. [48]

Diagnostic Utility:

Elevated PCT levels have been associated with bacterial infections, including CAP. Studies indicate that higher PCT concentrations correlate with increased severity of pneumonia in pediatric patients. For instance, children requiring hospitalization exhibited median PCT levels of 17.8 ng/mL, compared to 0.72 ng/mL in outpatients. Additionally, those admitted to intensive care units or with complications like empyema had higher median PCT levels, suggesting its potential to identify severe cases. [49]

Prognostic Value:

PCT levels have been linked to pneumonia severity and length of hospital stay (LOS). Higher PCT concentrations are associated with increased severity and longer LOS, indicating that PCT

may be useful in helping clinicians evaluate pneumonia severity. [50]

Guiding Antibiotic Therapy:

PCT-guided management has shown promise in reducing antibiotic exposure in pediatric patients with infectious diseases. By utilizing PCT levels to guide the initiation and duration of antibiotic therapy, unnecessary antibiotic use can be minimized, potentially reducing the risk of antibiotic resistance and other related complications. [51]

According to a study by Vinod H. Ratageri, radiographic pneumonia is substantially correlated with positive PCT (> 0.5 ng/mL), whereas WHO-defined pneumonia is not. On the other hand, it may serve as a stand-in for severe pneumonia.

Additional research by Rashmita Bora Although it cannot determine the severity of the disease in this context, PCT is a key diagnostic and prognostic marker of CAP. PCT's involvement in diagnosing and predicting pneumonia, particularly in adults, has been validated by many studies; however, its role in pediatric community-acquired pneumonia remains unclear. [52]

Limitations:

Despite its potential benefits, the use of PCT in pediatric CAP has limitations. Some studies have reported conflicting data regarding its diagnostic accuracy. For example, while PCT has shown higher predictive values in certain studies, other research has highlighted its limitations as a diagnostic aid for managing pediatric pneumonia. [53]

Conclusion:

Procalcitonin presents a valuable tool in the management of community-acquired pneumonia in children, offering insights into disease severity and aiding in antibiotic stewardship. However, clinicians should be aware of its limitations and consider it alongside clinical judgment and other diagnostic methods to ensure optimal patient care.

METHODOLOGY:

Source of Data:

Place of Study:

Paediatric ICU, Shri B. M. Patil Medical College Hospital and Research Centre, B.L.D.E. (Deemed University), Vijayapura, Karnataka

Duration of Study:

1.5 years (March 2023 – October 2024)

Study Design:

Prospective Cohort Study

Inclusion Criteria:

Children between the ages of two and fifty-nine months who exhibit fever, cough, hurried breathing and any of the following symptoms:

A quiet child's chest indrawing, stridor, grunting, lethargy, convulsions, and loss of appetite.

Exclusion Criteria:

The following children were excluded from the study if they:

- Had croup with evidence of a foreign body in the respiratory tract.
- Were already receiving antibiotics prior to admission at Shri. B.M. Patil Hospital, Vijayapura.

Method of Data Collection:

Informed Consent:

After obtaining informed consent, children meeting the inclusion criteria were admitted to the Paediatric ICU for evaluation.

History Collection:

A detailed clinical history was recorded to collect the information regarding:

- Duration of fever, cough, and hurried breathing.
- Past history of hospital admissions for similar complaints.

Clinical Examination:

Comprehensive clinical examination was performed for all the children:

- To measure the respiratory rate.
- To assess of chest retractions.
- Chest percussion and auscultation.

Observation and Evaluation:

The **Acute Illness Observation Scale (AIOS)** was used to assess the children on:

Day 1, Day 2 & Day 5 of admission.

Parameters with Scoring:

Observation item	1(Normal)	3(Moderate impairment)	5(Severe impairment)
Quality of cry	Strong with normal tone or content and not crying	Whimpering or sobbing	Weak or moaning or high pitched
Reaction to parent stimulation	Cries briefly then stops or content and not crying	Cries off and on	Continual cry or hardly responds
State variation	If awake, stays awake or if asleep and stimulated, wakes up quickly	Eyes close briefly awake or awakes with prolonged stimulation	Falls to sleep or will not rouse
Color	Pink	Pale extremities or acrocyanosis	Pale or cyanotic or mottled or ashen
Hydration	Skin and eyes normal and mucous membrane moist	Skin and eyes normal and mouth slightly dry	Skin doughy or tented & dry mucous membrane & or sunken eyes
Response (talk, smile) to social overtures	Smiles or alert(≤2mo)	Brief smile or alerts briefly(≤2mo)	No smile or face anxious, dull, expressionless or no alerting (≤2mo)

TABLE 1: AIOS Scale

Scale used	Acute illness observation scale
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Score interpretation	Each item scored as normal (=1)
	Moderate (=3) and severe
	Impairment (=5)
Total score	6= best score
	30= worst physical score

TABLE 2: AIOS Scoring

Laboratory Investigations:

On admission, 3ml of venous blood was drawn and sent for Serum Procalcitonin.

STATISTICAL ANALYSIS:

Sample size

With anticipated Mean ± SD of Bacterial pneumonia cases 12.0±6.7 , the study required a sample size of **47** patients with 95**%** level of confidence and a precision of 2

Formula used

$$n = \frac{z^2 S^2}{d^2}$$

Where Z = Z statistic at α level of significance

d^2 = Absolute error

S = Common standard deviation

Q = 100-p

STATISTICAL ANALYSIS

Data Analysis: The data collected was entered into a Microsoft Excel spreadsheet and analyzed using **Statistical Package for the Social Sciences (SPSS)**, Version 20.

Results Presentation:

- **Descriptive Statistics:**

Results are expressed as Mean (Median) ± Standard Deviation (SD), Interquartile range (IQR), Counts and percentages.

- **Graphical Representation:** Findings were illustrated using appropriate diagrams and charts.

RESULTS

Table 3: Age-wise distribution of children with Community-Acquired Pneumonia

Sl no	Variables	Frequency (n-51)	Percentages (%)
1	<12 months	24	47
2	12 months – 5 years	27	53
3	Total	51	100

This table shows age-wise distribution among children with community-acquired pneumonia and found that the majority were in the age group of 12 months -5years i.e., 53% (n-27) followed by 47.1% (n-24) in the age group of <12 months

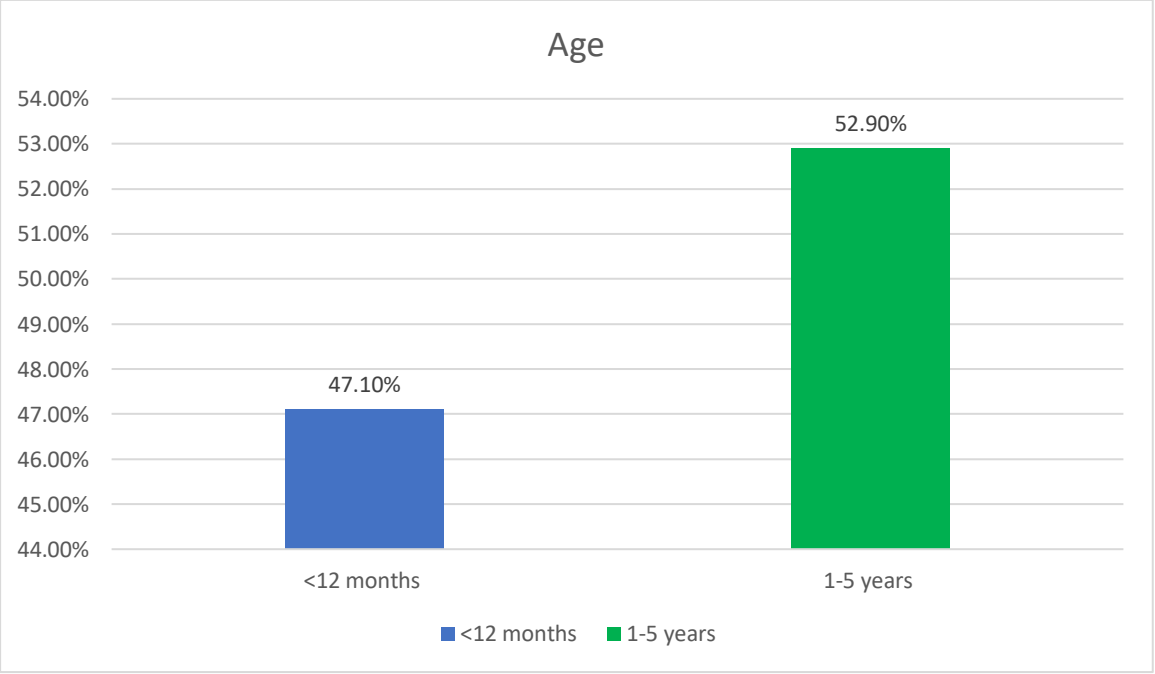

Figure 9: Bar diagram of Age-wise distribution of children with Community-Acquired Pneumonia

Table 4: Sex-wise distribution of children with community-acquired pneumonia

Sl no	Sex	Frequency (n-51)	Percentages (%)
1	Female	18	35
2	Male	33	65
3	Total	51	100

This table shows the distribution of Sex of the children with community-acquired and found that 65% (n-33) were male children and 35% (n-18) were female children and it is depicted in the pie chart.

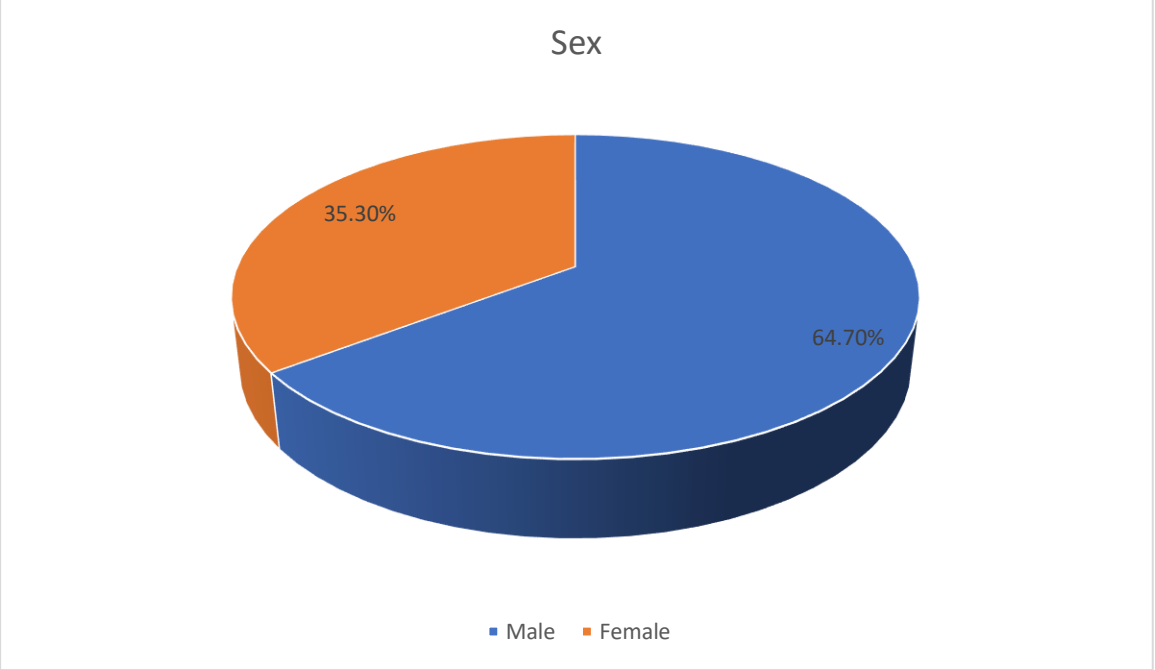

Figure 10: Distribution of sex among children with community-acquired pneumonia

Table 5: Distribution of children with community-acquired pneumonia according to Duration of fever

Sl no	Duration of fever	Frequency (n-51)	Percentages (%)
1	<5 days	46	90.2
2	>5 days	5	9.8
3	Total	51	100

This table shows the distribution of children with community-acquired pneumonia and the duration of fever and showed that 90.2% (n-46) had a fever of less than 5 days and only 9.8% (n-5) had a fever >5 days and it is shown in bar diagram who admitted due to fever.

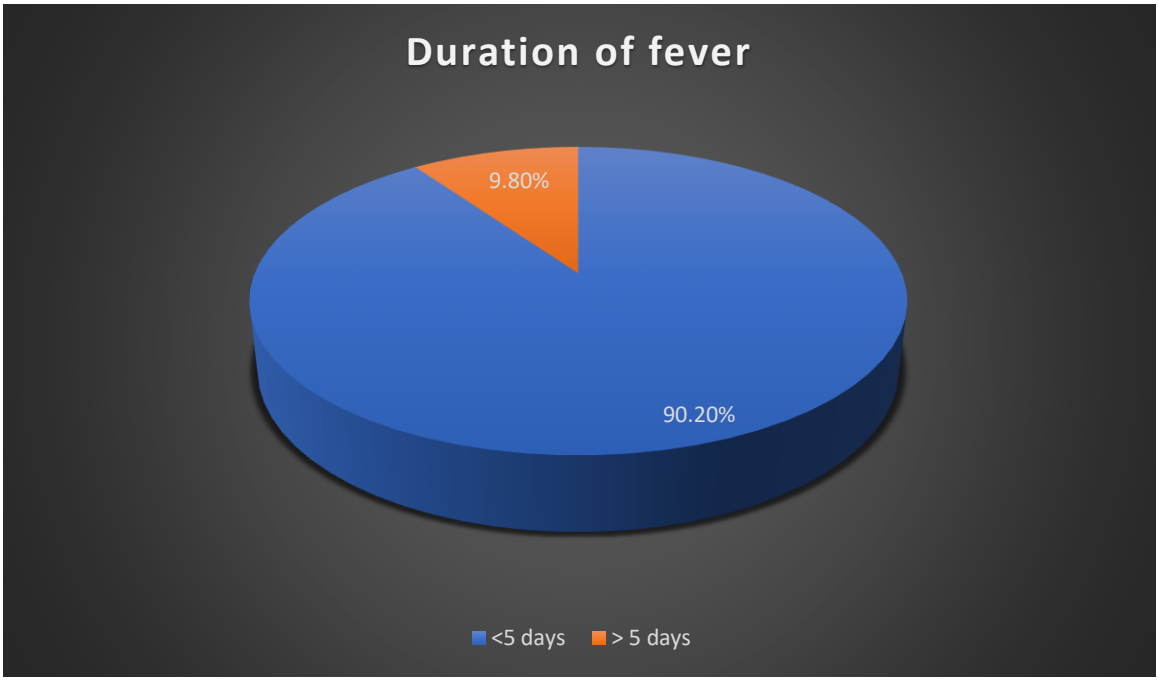

Figure 11: Distribution of children with community-acquired pneumonia according to Duration of fever

Table 6: Distribution of children with community-acquired pneumonia according to the Presence of cough

Sl no	Presence of cough	Frequency (n-51)	Percentages (%)
1	<7 days	43	84.3
2	>7 days	8	13.7
3	Total	51	100

This table shows the duration of cough among children with community-acquired pneumonia and showed that the majority of the children had a cough <7 days 84.3% (n-43) and only 13.7% had a cough duration >7 days.

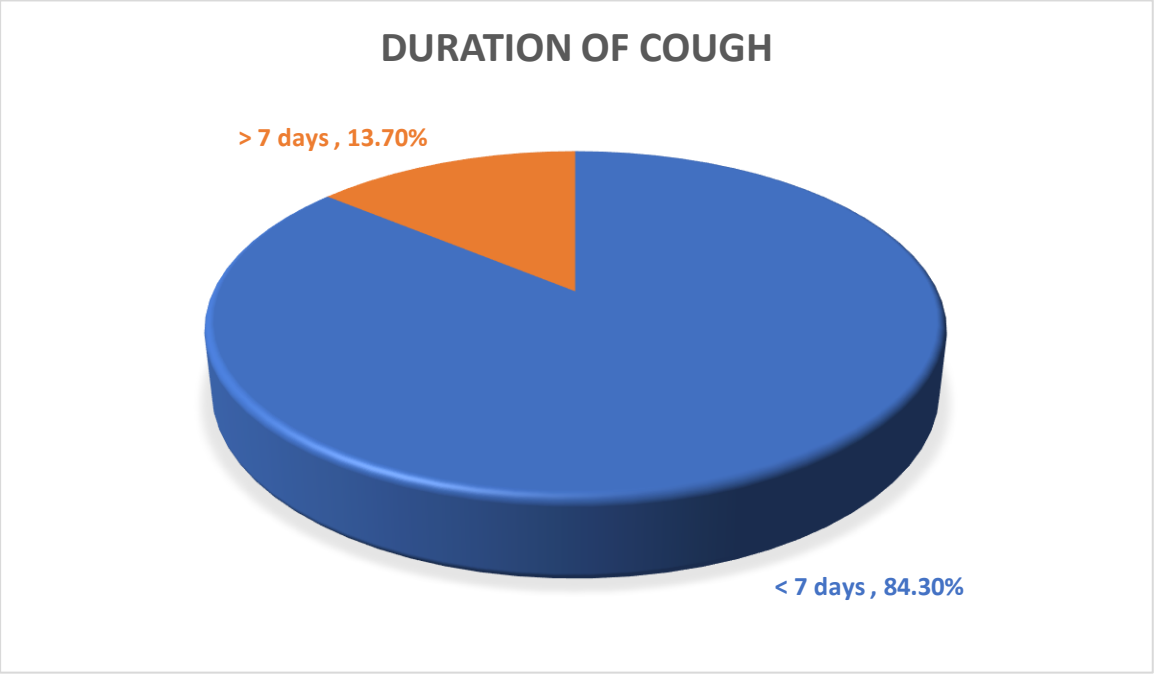

Figure 12: Distribution of children with community-acquired pneumonia according to the Presence of cough

Table 7: Distribution of Duration of hurried breathing among children with community-acquired pneumonia

Sl no	Duration of hurried breathing	Frequency (n-51)	Percentages (%)
1	1 day	35	68.6
2	2 days	14	27.5
3	3 days	2	3.9
4	Total	51	100

This table shows that majority (68.6%) of the children had hurried breathing for 1 day and 27.5% of children had it for 2 days and 3.9% had hurried breathing for 3 days.

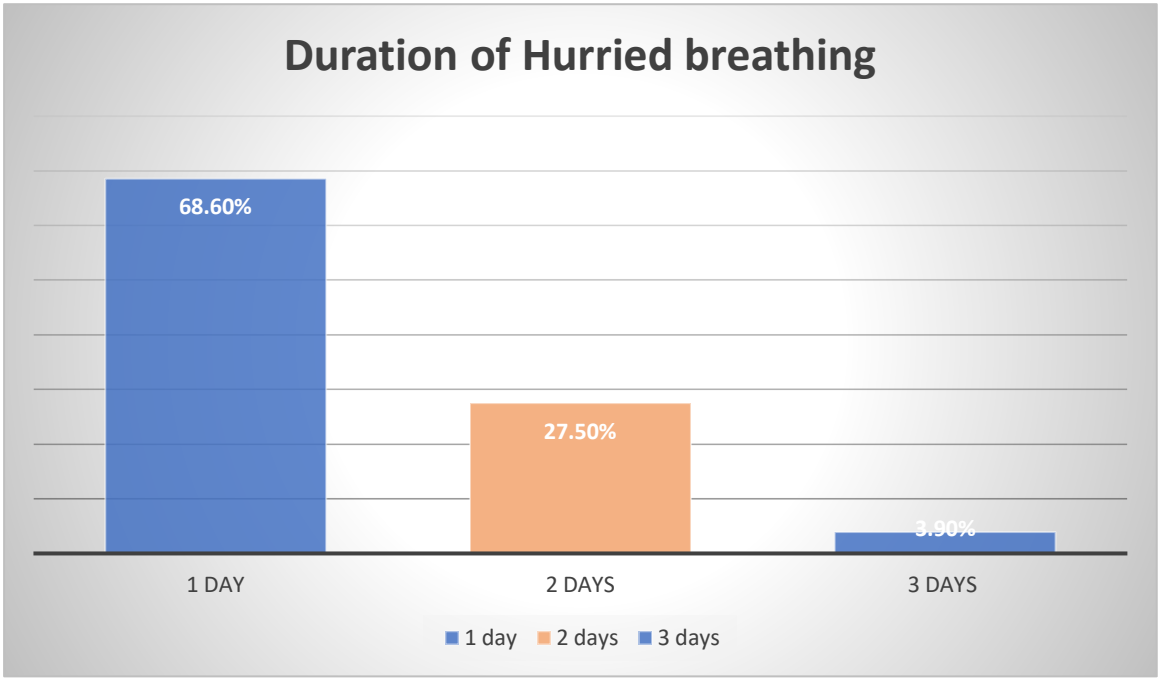

Figure 13: Distribution of Duration of hurried breathing among children with community-acquired pneumonia

Table 8: Distribution of Associated symptoms among children with community-acquired pneumonia

Sl no	Associated symptoms	Frequency (n-51)	Percentages (%)
1	Nil	35	69
2	Yes	16	32
3	Total	51	100

This table shows the children with associated symptoms along with community-acquired pneumonia (CAP) and found that 69% (n-35) had no Associated symptoms but 32% of children (n-16) had associated symptoms like vomiting, poor feeding, pain abdomen, etc.

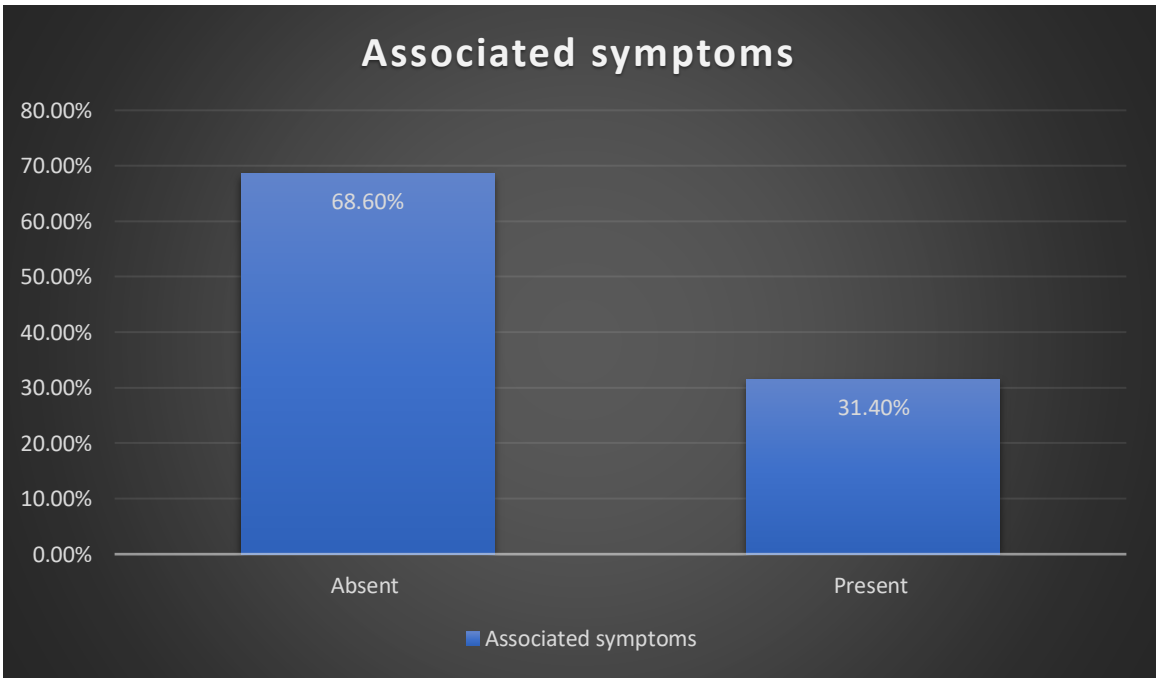

Figure 14: Distribution of Associated symptoms among children with community acquired pneumonia

Table 9: Distribution of Comorbidities among children with community-acquired pneumonia

Sl no	Comorbidities	Frequency (n-51)	Percentages (%)
1	Absent	36	70
2	Present	15	30
3	Total	51	100

This table shows the presence of comorbidities among children with CAP and found that 70.6% (n-36) of children with community-acquired pneumonia had no associated co-morbidities and 29.4% of children (n-15) had comorbidities like seizure disorder, febrile seizure, failure to thrive, etc.

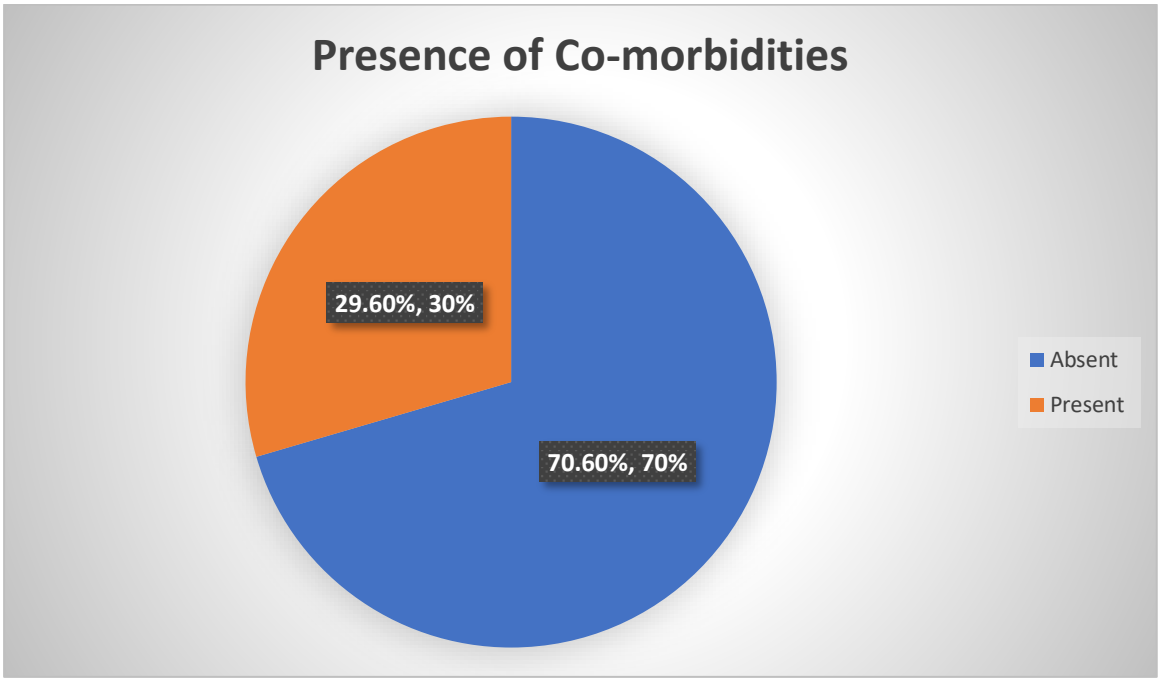

Figure 15: Distribution of Comorbidities among children with community-acquired pneumonia

Table 10: Distribution of past history and findings of general physical examination among children with community-acquired pneumonia

Variables		Frequency (n-51)	Percentages (%)
Past history (n-51)	Absent	30	59
	Present	21	41
General physical examination (n-51)	Absent	22	43
	Present	29	59

This table shows the Past history and general physical examination of children with community acquired pneumonia (CAP) and found that 41% (n-21) had similar complaints in the past few months and on general physical examination 59% (n-29) had some kind of signs like pallor, edema and cyanosis.

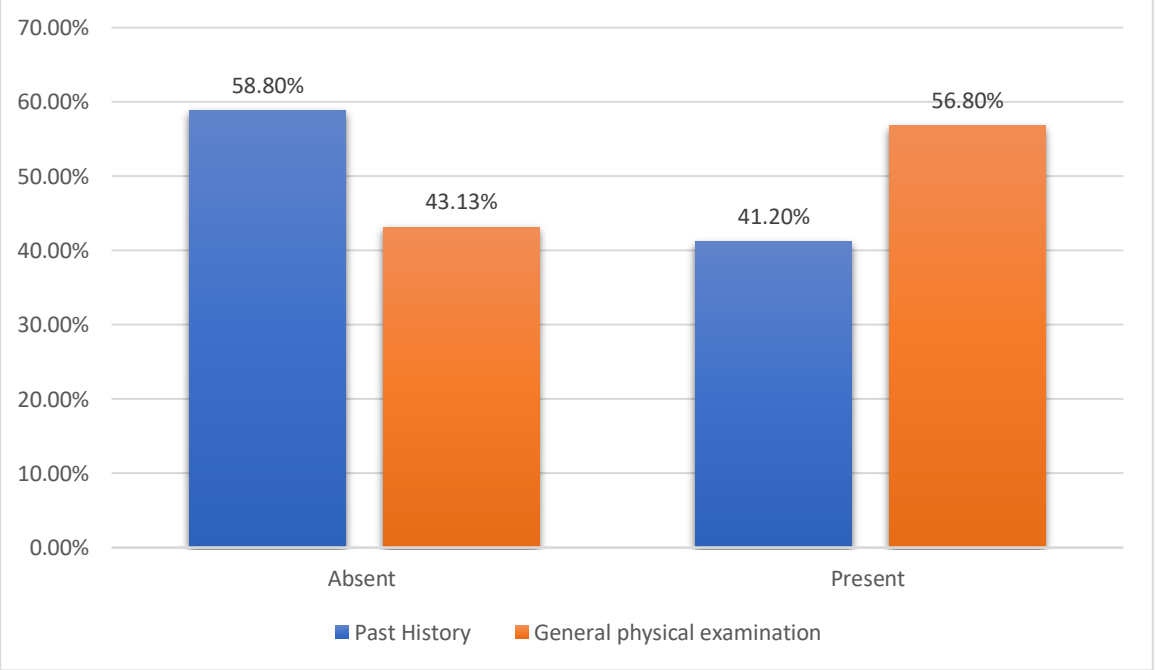

Figure 16: Distribution of past history and general physical examination among children with community-acquired pneumonia

Table 11: Distribution of Respiratory rate among children with community-acquired pneumonia

Sl no	Respiratory rate	Frequency (n-51)	Percentages (%)
1	Increased	24	47.1
2	Normal	27	52.9

This table shows the distribution of examination findings among children with community-acquired pneumonia. The respiratory rate was increased in 47.1% of children (24), according to age, and normal in 52.9% of children (n-27), as shown in the pie diagram.

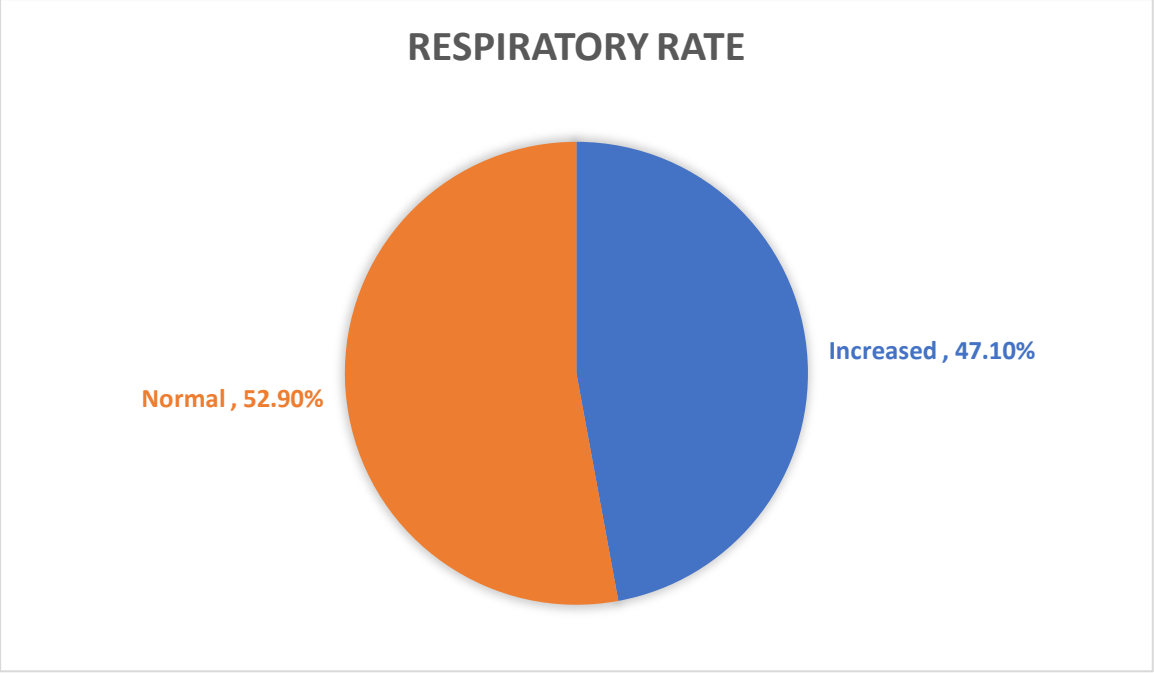

Figure 17: Distribution of Respiratory rate among children with community-acquired pneumonia

Table 12: Distribution of Grunting, Retraction Findings among children with community-acquired pneumonia

Variables		Frequency (n-51)	Percentages (%)
Grunt (n-51)	Absent	39	76.5
	Present	12	23.5
Retraction (n-51)	Present	31	61
	Absent	20	39

This table shows that on examination it was found that Retractions and Grunting was present in 61% and 23% of children with Community-acquired pneumonia respectively.

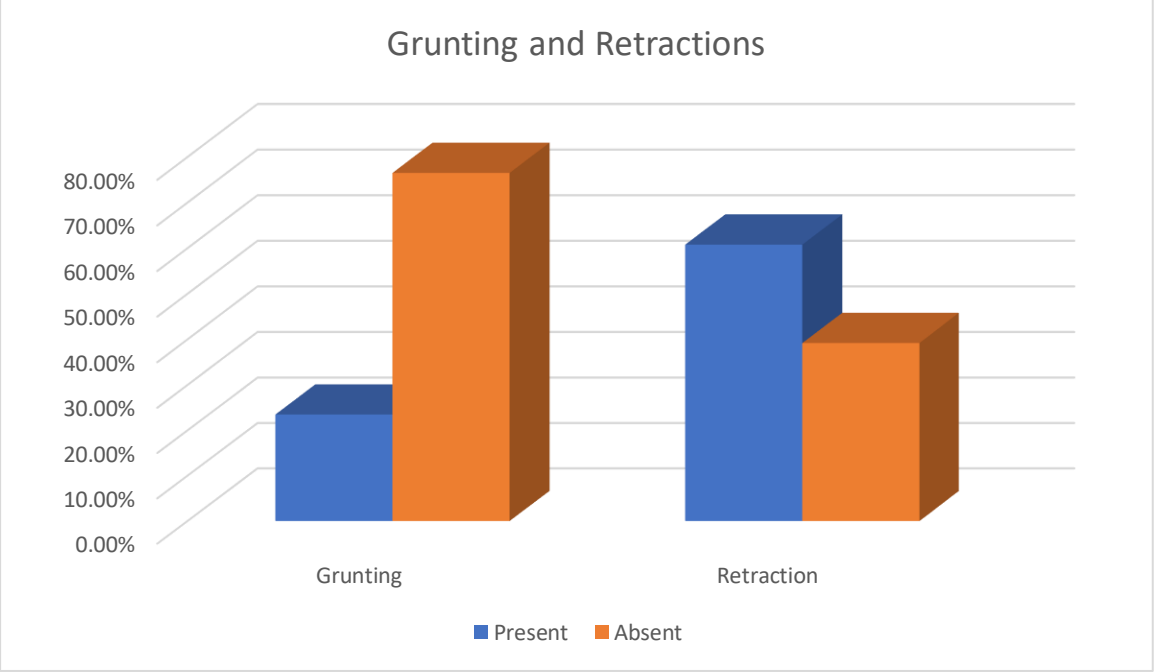

Figure 18: Distribution of Grunting, Retraction Findings among children with community acquired pneumonia

Table 13: Distribution of Examination findings among children with community-acquired pneumonia

Variables		Frequency (n-51)	Percentages (%)
Capillary refill time (n-51)	<3	44	86.3
	>3	7	13.7
Air entry (n-51)	Bilateral present	43	84.3
	Reduced	8	15.7
Added sounds (n-51)	Present	44	86.3
	Absent	7	13.7

This table shows examination findings of children with community acquired pneumonia and found that 86.3% (n-44) had added sounds and added sounds like Bilateral rhonchi, crepitations and bilateral air entry was reduced in only 15.7%.

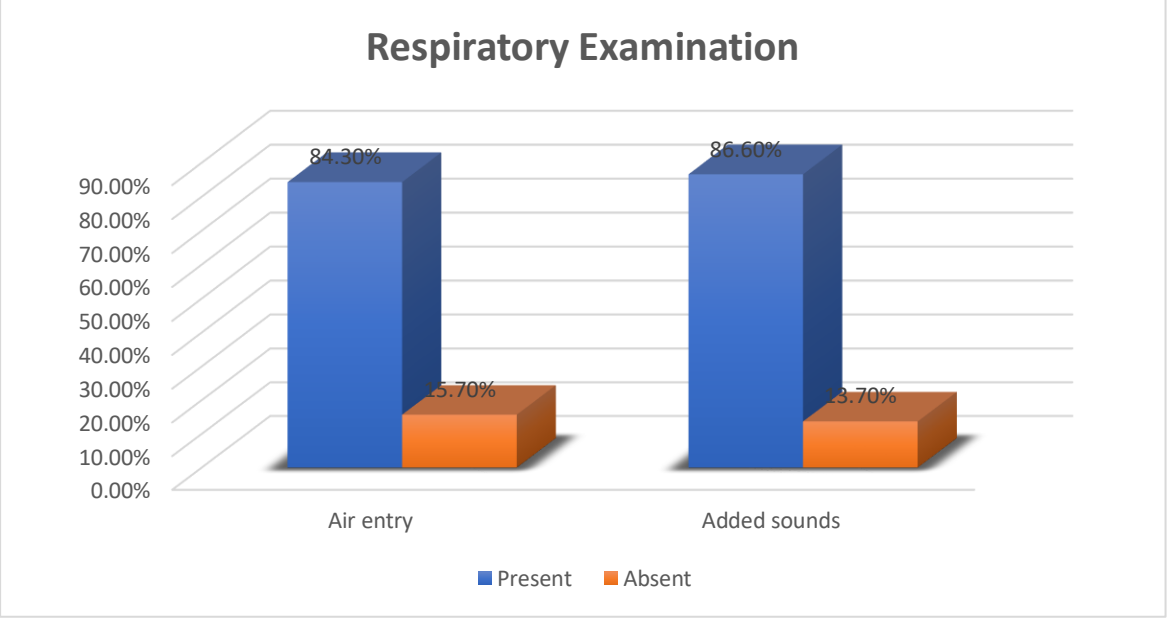

Figure 19: Distribution of Examination findings among children with community acquired pneumonia

Table 14: Distribution of CNS findings among children with community acquired pneumonia

Sl no	CNS	Frequency (n-51)	Percentages (%)
1	Normal	47	92.2
2	CNS changes	4	7.8
3	Total	51	100

This table shows that on examination of CNS 92.2% of children were alert and 5.9% were irritable and drowsy.

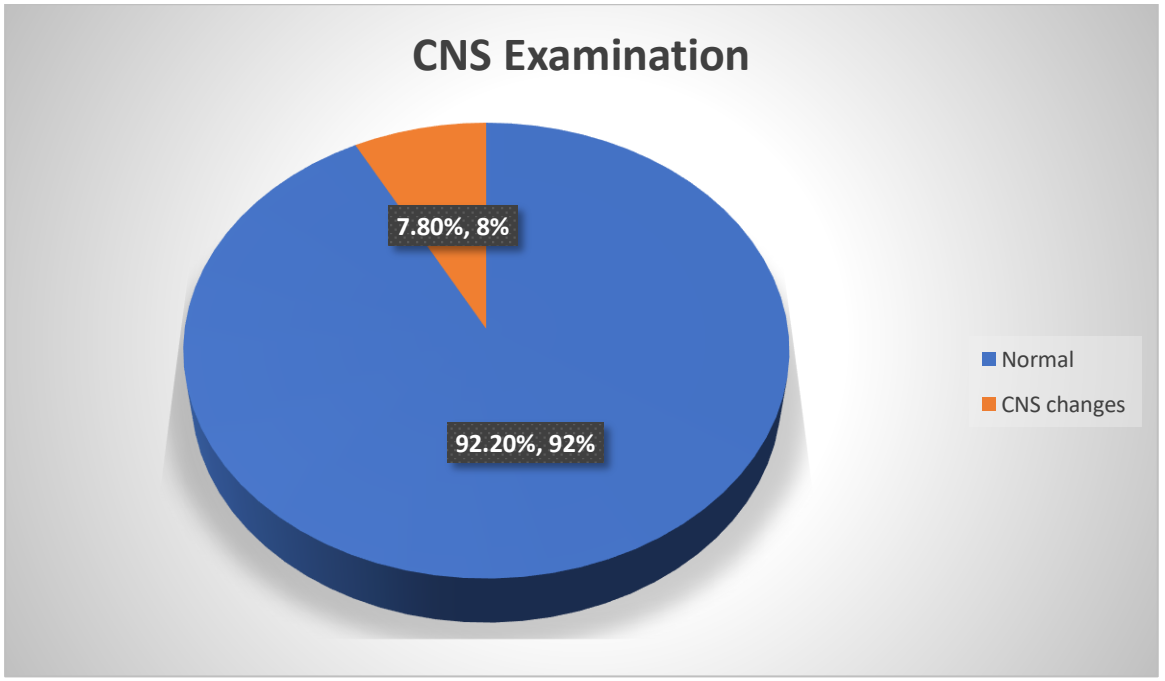

Figure 20: Distribution of CNS findings among children with community-acquired pneumonia

Table 15: Distribution of Various treatment modalities among children with community-acquired pneumonia

Variables		Frequency (n-51)	Percentages (%)
Oxygen Requirement (n-51)	Yes	50	98.0
	NO	1	2.0
Respiratory Support (n-50)	High flow nasal cannula (HFNC)	26	52
	Ventilation	7	14
	Nasal Prongs	17	34
Surgical procedure (n-51)	Yes	5	9.8
	No	46	90.2

This table shows the distribution of requirement of oxygen level and found that 98% need oxygen and 51% (n-26) and the respiratory supports required were HFNC for 33.3% (n-17) and nasal prongs, whereas 13.7% required ventilation (n-7) and only 9.8% (n-5) had surgical procedure like bilateral ICD insertion.

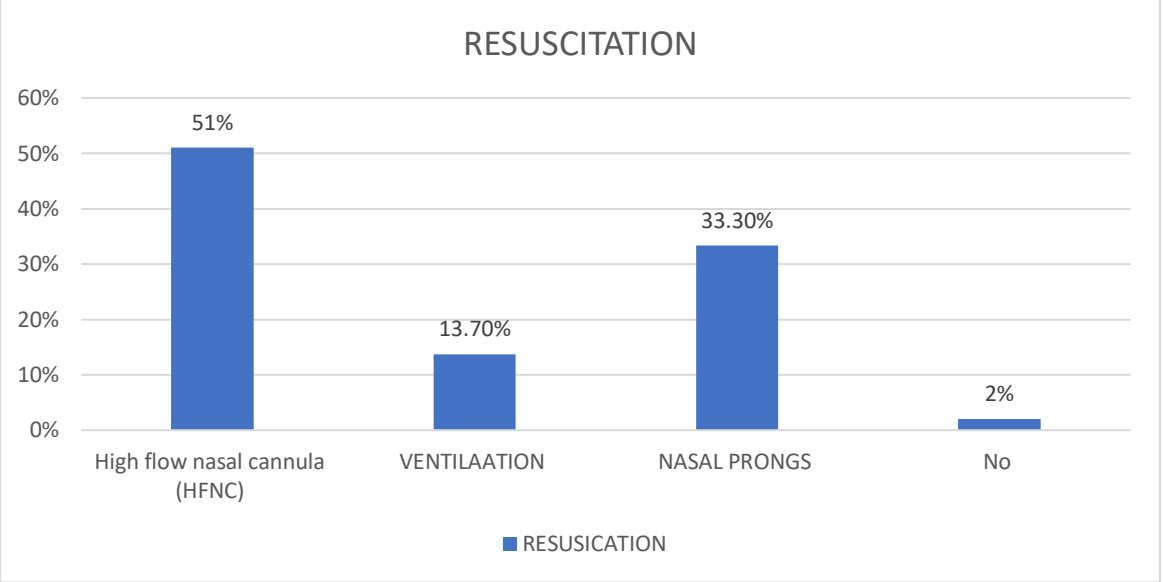

Figure 21: Distribution of Various treatment modalities among children with community-acquired pneumonia

Table 16: Distribution of AIOS scale among children with community-acquired pneumonia

AIOS scale on day wise		Frequency (n-51)	Percentages (%)
AIOS ON DAY 1 (n-51)	Mild	14	27.5
	Moderate	29	56.9
	Severe	8	15.7
AIOS ON DAY 2 (n-51)	Mild	32	62.7
	Moderate	8	15.7
	Severe	10	19.6
AIOS ON DAY 5 (n-51)	Mild	37	72.5
	Moderate	2	3.9
	Severe	2	3.9

This table shows the Acute illness observation scale for children with community acquired pneumonia and found that 56.9% (n-29) were having moderate AIOS on day 1 and then 62.7% (n-32) had Mild AIOS on day 2 followed by 72.5% (n-37) had mild on AIOS on day 5.

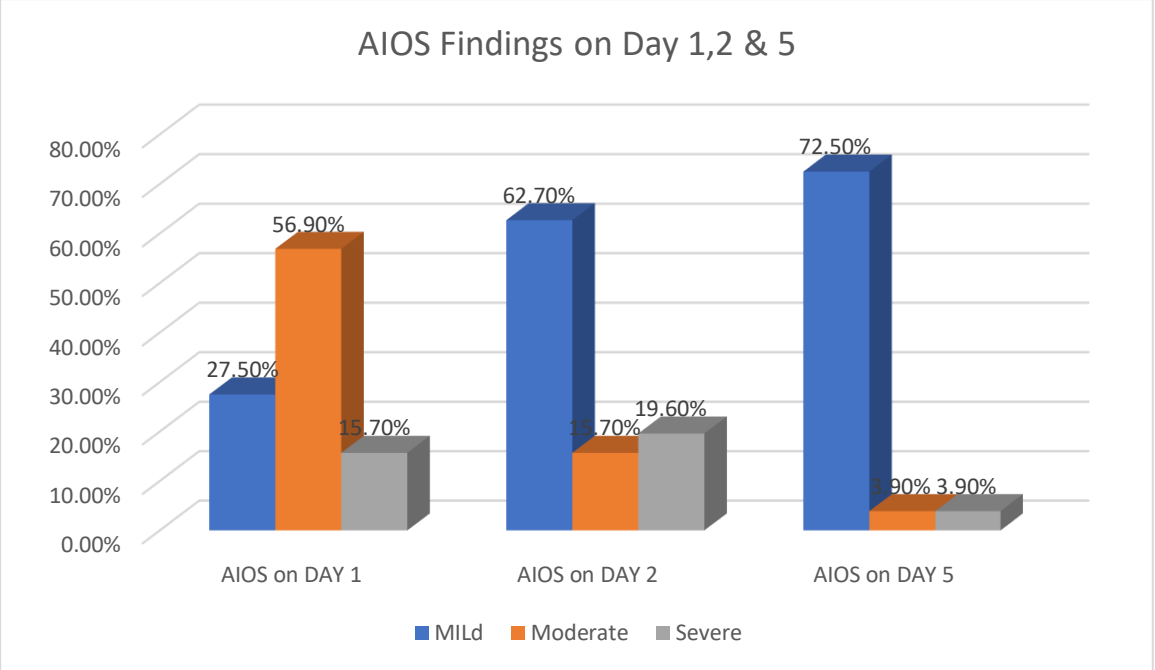

Figure 22: Distribution of AIOS findings on Days 1, 2, and 5 among children with community-acquired pneumonia

Table 17: Distribution of CRP gradings among children with community-acquired pneumonia

Sl no	CRP	Frequency (n-51)	Percentages (%)
1	I	16	31.4
2	II	11	21.6
3	III	8	15.7
4	IV	16	31.4

This table shows CRP values among children with community acquired pneumonia and found that 31.4% (n-16) had grade I and grade IV CRP followed by 21.6% (n-11) had grade II CRP and it is shown in pie diagram.

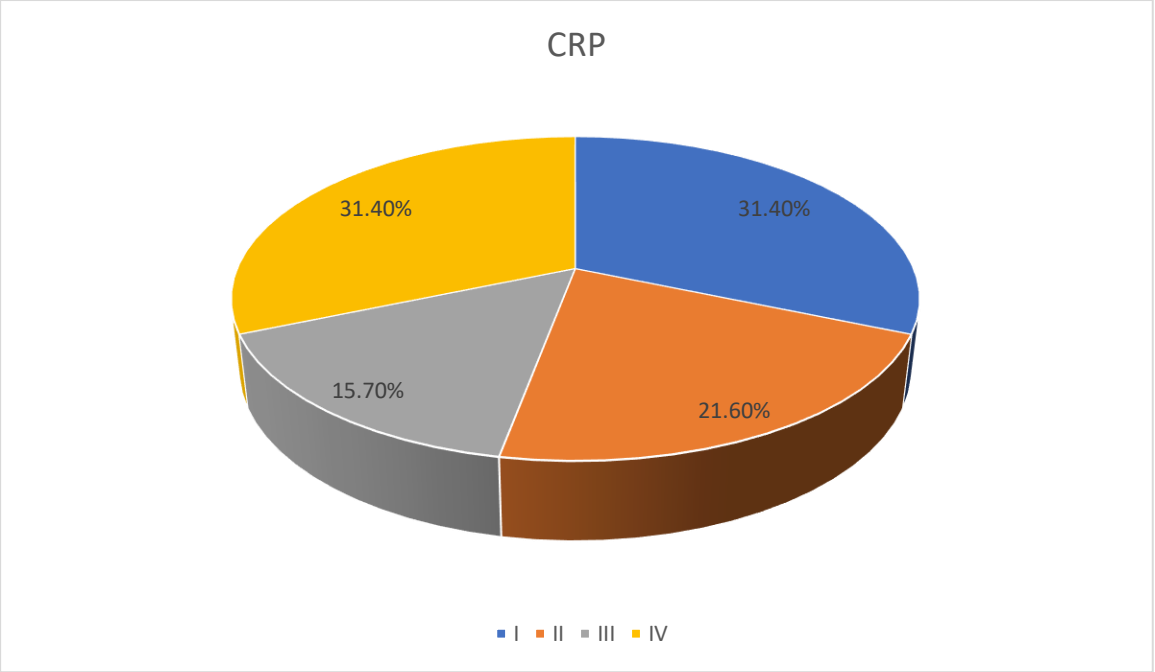

Figure 23: Distribution of CRP gradings among children with community-acquired pneumonia

Table 18: Distribution of Outcome results among children with community-acquired pneumonia

Sl no	outcome	Frequency (n-51)	Percentages (%)
1	Improved	44	86.3
2	Dead	7	13.7

This table shows the outcome results among children with community-acquired pneumonia and found that out of 51 children 13.7% (n-7) of children died due to complications.

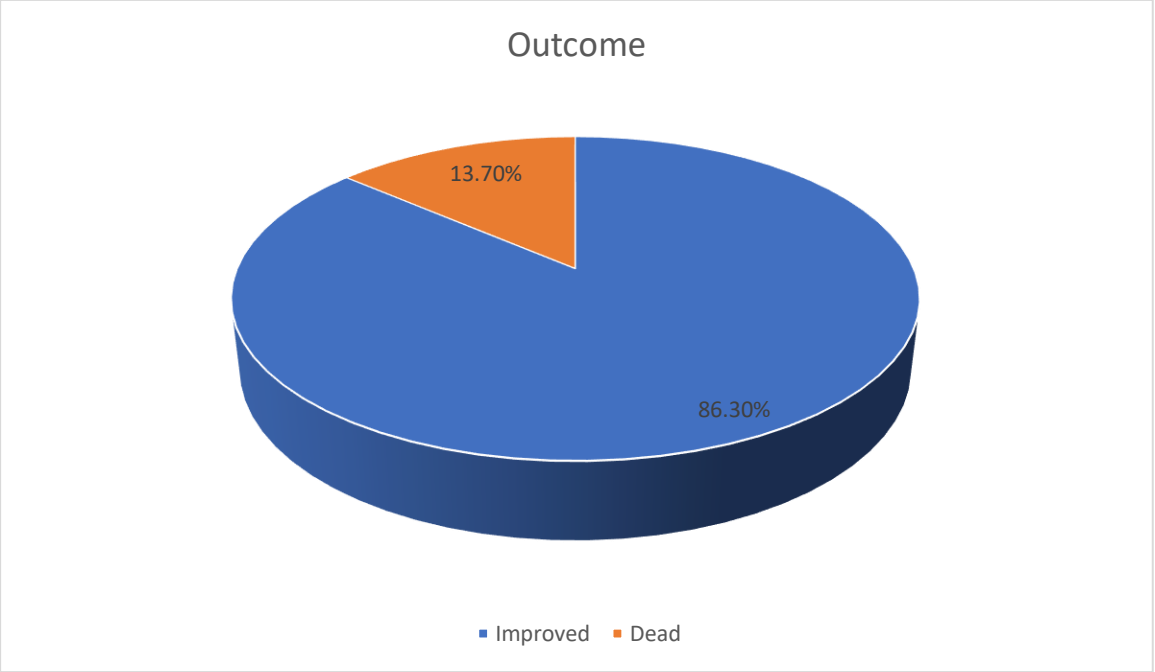

Figure 24: Distribution of Outcome among children with community-acquired pneumonia

Table 19: Association between Sociodemographic Profile and AIOS Scale on Day 1

Variables	AIOS on day 1			P value
Age	**Mild**	**Moderate**	**Severe**	P value-0.630
	8(33.3%)	13(54.2%)	3(12.5%)	X^2= 0.923
	6(22.2%)	16(59.3%)	5(18.5%)	Df-2
Sex	6(33.3%)	8(44.4%)	4(22.2%)	P value-0.394
	8(24.2%)	21(63.6%)	4(12.1%)	X^2= 1.86
				Df-2

This table shows an association between Sociodemographic details and the AIOS Scale on day 1 and found that there is no association between Age and sex and the AIOS scale and the P value was found to be p value >0.05

Table 20: Association between symptoms and AIOS Scale on Day 1

Variables	AIOS on day 1			Total	P value
	Mild	Moderate	Severe		
Fever	12(26.1%)	27(58.7%)	7(15.2%)	46(100%)	P value -0.719
	2(40.0%)	2(40.0%)	1(20.0%)	5(100%)	X^2= 0.661
					Df-6
Cough	11(25.6%)	26(60.5%)	6(14.0%)	44(100%)	P value-0.281
	3(42.9%)	2(28.6%)	2(28.6%)	7(100%)	X^2= 2.531
Duration of Hurried Breathing (in days)	10(28.6%)	19(54.3%)	6(17.1%)	35(100%)	P value - 0.801
	4(28.6%)	8(57.1%)	2(14.3%)	14(100%)	X^2= 1.646
	0.0%	2(100.0%)	0.0%	2(100%)	
Symptoms	10(28.6%)	20(57.1%)	5(14.3%)	35(100%	P value - 0.908
	4(25.0%)	9(56.2%)	3(18.8%)	16(100%)	X^2= 0.303
Co- morbidities	10(27.8%)	21(58.3%)	5(13.9%)	36(100.0%)	P value - 0.859
	4(26.7%)	8(53.3%)	3(20.0%)	15(100.0%)	X^2= 0.303
Past history	6(20.7%)	17(58.6%)	6(20.7%)	29(100.0%)	P value -0.209
	8(38.1%)	11(52.4%)	2(9.5%)	21(100.0%)	X^2= 3.3

This table shows the association between Symptoms od community-acquired pneumonia and AIOS scale measurement among children suffering from Community-acquired pneumonia and found that there is no association between various symptoms and AIOS scale and the p-value was found to be not significant.

Table 21: Association between Respiratory examination findings and AIOS Scale on Day 1

Sl no	AIOS on day 1				Total	P value
		Mild	Moderate	Severe		
Respiratory rate	Increased	9(37.5%)	11(45.8%)	4(16.7%)	24(100.0%)	P value-0.264 X^2= 2.66
	Normal	5(18.5%)	18(66.7%)	4(14.8%)	27(100.0%)	
Grunting	Absent	14(35.9%)	21(53.8%)	4(10.3%)	39	P value-**0.021** X^2= 7.64
	Present	0.0%	8(66.7%)	4(33.3%)	12	
Retraction	Present	3(11.1%)	17(63.0%)	7(25.9%)	27(100.0%)	P value-**0.07** X^2= 9.9
	Absent	10(50.0%)	9(45.0%)	1(5.0%)	20(100.0%)	
Air entry	Bilateral present	13(30.2%)	23(53.5%)	7(16.3%)	43(100.0%)	P value-0.501 X^2= 1.38
	Decreased	1(12.5%)	6(75.0%)	1(12.5%)	8(100.0%)	
Added sounds	Present	12(27.3%)	25(56.8%)	7(15.9%)	44(100.0%)	P value-0.993 X^2= 0.014
	Absent	2(28.6%)	4(57.1%)	1(14.3%)	7(100.0%)	
Surgical procedure	Yes	0.0%	4(80.0%)	1(20.0%)	5(100.0%)	P value-0.348 X^2= 2.56
	No	14(30.4%)	25(54.3%)	7(15.2%)	46(100.0%)	

This table shows the association between AIOS scale on day 1 and respiratory examination findings and it was found on applying chi-square test Grunting and retraction is associated with AIOS on day 1 and p value found to be statistically significant (p value <0.05).

Table 22: Association between CRP VALUE and AIOS Scale on Day 1

Sl no	AIOS on day 1			Total	P value
CRP	Mild	Moderate	Severe		
I	8 (50.0%)	7 (43.8%)	1 (6.2%)	16 (100.0%)	**0.0030**
II	3 (27.3%)	5 (45.5%)	3 (27.3%)	11 (100.0%)	
III	3 (37.5%)	3 (37.5%)	2 (25.0%)	8 (100.0%)	
IV	0 0.0%	14 (87.5%)	2 (12.5%)	16 (100.0%)	

X^2= 13.950,6, DF - 9

This table shows the association between CRP level and AIOS on day 1 and found that after applying chi-square test, p value **found to be statistically significant** (p value<0.05).

Table 23: Association between CRP VALUE and AIOS Scale on Day 2

Sl no	AIOS on day 2			Total	P value
CRP	Mild	Moderate	Severe		
I	12 (75.0%)	3 (18.8%)	1 (6.2%)	16 100.0%	0.192
II	8 (72.7%)	0 0.0%	3 (27.3%)	11 (100.0%)	
III	3 (37.5%)	1 (12.5%)	4 (50.0%)	8 (100.0%)	
IV	10 (56.2%)	4 (25.0%)	2 (12.5%)	16 (100.0%)	

X^2= 13.39, DF - 9

This table shows the association between CRP level and AIOS on day 2 and found that after applying chi-square test, p value found to be not significant (p value >0.05).

Table 24: Association between serum procalcitonin level and AIOS Scale on Day 1

Serum procalcitonin	AIOS on day 1			Total	P value
	Mild	Moderate	Severe		
I	6 (50.0%)	4 (33.3%)	2 (16.7%)	12 (100.0%)	**0.021**
II	6 (46.2%)	6 (46.2%)	1 (7.7%)	13 100.0%	
III	1 (11.1%)	8 (88.9%)	0 0.0%	9 (100.0%)	
IV	1 (5.9%)	11 (64.7%)	5 (29.4%)	17 (100.0%)	

X^2= 14.86, DF - 9

This table shows association between serum procalcitonin level and AIOS on day 1 and subjected to statistical test and Fisher's Exact test was used and p value was found to be <0.05 and found to be **statistically significant**.

Table 25: Association between serum procalcitonin level and AIOS Scale on Day 2

	AIOS on day 2			Total	P value
Serum procalcitonin	**Mild**	**Moderate**	**Severe**		
I	8(66.7%)	3(25.0%)	1(8.3%)	12(100.0%)	**0.053**
II	12(92.3%)	0(0.0%)	1(7.7%)	13(100.0%)	
III	7(77.8%)	1(11.1%)	1(11.1%)	9(100.0%)	
IV	1(5.9%)	5(29.4%)	4(23.5%)	7(41.2%)	

X^2= 16.78, DF – 9

This table shows the association between serum procalcitonin level and AIOS on day 2 and found that after applying chi-square test, p value **found to be statistically significant** (p value <0.05).

Table 26: Association between Duration of stay in the hospital and AIOS Scale on Day 1

Sl no	AIOS on day 1			Total	P value
Stay	**Mild**	**Moderate**	**Severe**		
1	10 (33.3%)	16 (53.3%)	4 (13.3%)	30 (100.0%)	5.13
2	4 (19.0%)	13 (61.9%)	4 (19.0%)	21 (100.0%)	

X^2= 1.335, DF – 6

This table shows association between Duration of stay in the hospital and AIOS on day and subjected to statistical test and Fisher's Exact test was used and p value was found to be >0.05 and it was found that there was no significant association between duration of hospital stay and AIOS.

Table 27: Association between Outcome and AIOS Scale on Day 1

Outcome	AIOS on day 1			Total	P value
	Mild	**Moderate**	**Severe**		
Discharged	14 (31.8%)	27 (61.4%)	3 (6.8%)	44 (100.0%)	**0.05**
Death	0 0.0%	2 (28.6%)	5 (71.4%)	7 (100.0%)	

X^2= 19.45, DF – 4

This table shows the association between outcome and AIOS on day 1 and found that after applying chi-square test, p value **found to be statistically significant** (p value <0.05).

Table 28: Association between Outcome and AIOS Scale on Day 2

Outcome	AIOS on day 2			Total	P value
	Mild	Moderate	Severe		
Discharged	32 72.7%	8 18.2%	4 9.1%	44 100.0%	**0.01**
Death	0 0.0%	0 0.0%	6 85.7%	7 100.0%	

X^2= 30.7, DF – 4

This table shows the association between outcome and AIOS on day 1and found that after applying chi-square test, p value **found to be significant** (p value <0.05).

Table 29: Association between Serum procalcitonin and CRP values

	Serum procalcitonin				Total	P value
CRP	**I**	**III**	**III**	**IV**		
I	11(68.8%)	2(12.5%)	0(%)	3.18.8(%)	16.(100%)	**0.01**
II	1(9.1%)	8(72.7%)	1(9.1%)	1(9.1%)	11(100%)	
III	0(%)	2(25%)	1(12.5%)	5(62.5%)	8(100%)	
IV	0	1(6.2%)	7(43.8%)	8(50%)	16(100%)	

X^2= 49.4, DF – 9

This table shows the association between CRP values and serum procalcitonin and it **found to statistically associated** and p value found to be significant (<0.01).

DISCUSSION:

One of the most prevalent infectious diseases in underdeveloped nations is undoubtedly childhood pneumonia, which also contributes significantly to avoidable child mortality. One of the most prevalent illnesses among children is community-acquired pneumonia (CAP). Since it is the leading cause of hospitalization and the second leading cause of death for children in underdeveloped nations, it has been the subject of much research.

McCarthy PL et al. conducted a study in 1982 to determine whether systematic observational evaluation improves the effectiveness of the traditional history-taking and physical examination method in identifying serious disease in children with fever. To determine the sensitivity of the combined examination, he looked into children less than 24 months who had fever. This study showed that the combination of AIOS scoring, history, and physical examination had a better sensitivity and association for serious disease than the normal history and physical examination alone.

AIOS helps recognize children who are feverish yet have a serious disease, as McCarthy et al. have previously shown. [7] Our study demonstrated that when history and examination are integrated with AIOS scoring, the sensitivity and association for serious illness are higher than with standard history taking and physical examination. Thus, the goal of this study was to score the child's condition and evaluate the child using the criteria of the Acute Illness Observation Scale.

According to our study titled, Study of Acute Illness Observation Scale and Serum Procalcitonin Levels in Community Acquired Pneumonia in Children Aged 2 months to 59 months, a cross-sectional study was conducted to determine the relationship between the AIOS score and the clinical characteristics of pneumonia, the need for oxygen therapy, the length of

hospital stay, and the need for intensive care unit admission. The study involved 51 children, ages 2 to 59 months, who were admitted to the paediatric department from March 2023 to October 2024 for pneumonia after their primary caregiver gave their informed consent. 52.9% of the people in the 1–5-year age group were in this age group, while 47.1% were in the <12-month age group and 64.7% of them were male children.

A study by **Vidhusree et al.,** revealed a male preponderance of pneumonia (55.9% vs. 44.1%) similar to our study. The mean age of female children was slightly higher than that of male children (22.4±16.1 months against 23.3±15.7 months). Pneumonia was more common in older infants (24.8% in the 6 -11-month group), however, beyond the age of two, the frequency significantly decreased [54]. In terms of IMNCI classification, 27.3% of the study population had severe pneumonia, 14.7% had danger symptoms, 26.1% had chest indrawing, and 8% had stridor.

Chinchu Mariyam et al.'s study revealed that 203 individuals, or 56.4% of the total, were between the ages of 2 and 12 months. This age group had noticeably higher AIOS scores.[55] the same observation was noticed in our study. Awasthi et al. conducted a study in Lucknow that found that infants aged 2–11 months had a CAP incidence that was almost five to ten times greater than that of infants aged 12–59 months. [6] Incidence was similar in our study.

We found that out of 51 children, 90.2% had fever and were admitted for <5 days, and 84.3% were admitted for cough for <7 days, 98.6% had hurried breathing for 1 day, and 31.4% had some kind of associated symptoms like vomiting, poor feeding, and pain abdomen.

All children who presented with fever, cough, and dyspnoea were included in the study by **Aarthy et al.,** however, the key indicators for establishing a clinical diagnosis of pneumonia were tachypnoea (100%) and chest retractions (60%). [56] Tachypnoea, accounts for 74% sensitivity and a 67% specificity for radiologically diagnosed pneumonia, according to another

study by Palafox et al. Twenty percent of the children admitted for the research had pneumonia, while the remaining eighty percent had severe pneumonia, according to IMNCI. [57]

According to our study, 47.1% had an elevated respiratory rate, 60.78% had retraction, 23.5% grunted, and 86.3% had additional sounds including crepitation and rhonchi. These results were comparable to research conducted by **Chinchu Mariyam**, which revealed that drowsiness was the most prevalent risk indicator, followed by incapacity to drink, convulsions, and stridor, in that order.

Lethargy was the most common risk indication (32.1%), followed by convulsions (4.6%) and grunting (2.8%), according to another study by Muruli conducted in Bangalore. [58] According to a different study on AIOS by Lingaraj, 88.9% and 80% of kids received normal colour and hydration scores. [59]

A study by **Arathy et al**. revealed that children who scored higher than 16 at the time of admission exhibited lethargy, grunt (20%), and crepitations (20%) as additional important criteria. which was comparable to our research Additionally, the **Bharti** [6] study on the role of AIOS in treating severe pneumonia revealed that children who scored higher than 16 at the time of admission had a considerably higher percentage of children with tachypnoea, retractions, and grunts (55.5%, 55.5%, respectively).

Lethargy, cyanosis, grunting, chest retractions, tachypnoea, tachycardia, and crepitations on auscultation were all significantly associated with hypoxemia ($p<0.05$), according to a study by **Abhitej Reddy**. Similar to our research, 31 shows a significant correlation between retractions and grunting ($p <0.005$).

Our study revealed that 29.4% of the 51 children with community-acquired pneumonia had some form of comorbidities, such as seizure disorders, failure to grow, and febrile seizures. Of those with capillary refill time <3 minutes, 86.3% showed cyanosis, oedema, and pallor in 56.8% of cases.

These results were consistent with research by **Chinchu Mariyam et al,** that found a significant difference in participants whose capillary refill time was longer than two seconds. The AIOS score was more than 15 for 43 (97.7%) of the individuals whose capillary refill time was longer than 2 seconds.

To evaluate the AIOS scoring system's effectiveness in treating severe paediatric pneumonia in patients aged 2 to 59 months, **Bhavneet Barathi et al., [6]Prerena et al., and Akash Bang et al.** used this score to predict bacteraemia in feverish 36-month-old children. [59,60] The sensitivity and specificity of the AIOS score in predicting the prognosis of children with pneumonia in the age range of 2 to 59 months were examined by **Murali B.H. et al.** by comparing it with the IMCI.[58]

In the current study, we discovered that, on day 1, 27.5% of the 51 children had mild AIOS, or a score of less than 10, followed by 56.9% with a score of 11–15 (moderate), and 15.7% with a score greater than 16. We discovered that age, sex, and other pneumonia symptoms and AIOS do not significantly correlate. According to a different study by **Anoop K**, 40% of children with community-acquired pneumonia had aberrant initial evaluation scores (AIOS>10). It is evident from the mean score of AIOS 12.32 (SD-6.12) that all of the youngsters involved in the study are taken seriously.[4]

According to identical results, the research population's AIOS score ranged from 6 to 26 in another study by **Vidhushree et al** [54.] There were the most children in the well-looking group (39.1%, 93/238) and the most children in the seriously-ill-looking group (28.2%, 67/238), with the greatest number of children (23.1%, 55/238) having an AIOS score of 12.

The first line of antibiotics was used to treat all of the children with CAP, in contrast to a study by **Anoop K et al**, which found that 50.4% (125/248) of kids received parenteral antibiotics, while the remainder of the patients received oral drugs.

We found that 98% of children needed oxygen therapy, 51% needed a high flow nasal cannula, and 13.7% needed ventilation support. These findings were similar to a study by Arathy et al. that found that roughly 66% of children needed nasal oxygen due to respiratory distress, with 18/33 of those children having an AIOS score greater than 16.

Everyone who was admitted had a complete blood count and blood culture, and children with AIOS >16 had a significant statistical correlation for leukocytes ($p<0.05$), which is comparable to the findings of the study by **Aarathy et al.**

Our research revealed that the AIOS score improved significantly from day 1 to day 5. On day 1, the AIOS score was 27.5%, which was less than 10. On day 2 and day 5, the score improved to 62.7% and 72.5%, respectively. Similar findings were found in the study by Arathy et al. On the first day, 38% of patients received a score of 16, and on the fifth day, none of the patients received a score greater than 16. The reduction in the score between day 1 and day 5 was statistically significant among all the three scores ($p<0.005$)[56]

A study by **David T et al** [61] showed that testers should value results at the same level as fundamental symptoms and accepted clinical indicators and prevent lab results from being detached from clinical evaluations which was first documented one hundred years earlier. The

value of laboratory results matches that of cardinal symptoms and clinical signs which shows why they should merge with clinical observations to make proper diagnoses possible. The diagnostic accuracy of procalcitonin (PCT) testing remains average when used independently but its strategic association with clinical risk evaluations improves the accuracy of risk predictions for community-acquired pneumonia. A Clinical prediction rules have two significant limitations, though: they can be applied incorrectly or forgotten by physicians, and there can be a wide range of results within a given risk category. [62]

The most commonly researched conventional biomarker of infection is CRP. The liver produces this acute phase protein in reaction to IL-6. After an inflammatory stimulation, its blood concentration starts to increase 4–6 hours later, doubles every 8 hours, and peaks 36–50 hours later, with a half-life of 19 hours. Neutropenia, systemic steroids, and RRT are likewise believed to not affect its concentration. The low cost of the CRP test makes it very helpful for facilities with less funding. When compared to healthy controls, CRP is higher in patients with community-acquired pneumonia and can differentiate between pneumonia and heart failure and COPD exacerbations. A decrease in CRP is linked to recovery and a better prognosis in individuals with a serious infection. Using CRP point-of-care testing to direct antibiotic treatment in the primary care context.[63]

Although procalcitonin's significance in infections in newborns and children is still unclear, it is well-established as a biomarker for sepsis. CAP is one of the most prevalent infections in children and has been the subject of comprehensive research. An attempt was made in this study to determine whether procalcitonin and pneumonia are related.

Out of 51 study participants, we discovered that 33.3 (n = 17) had grade IV procalcitonin, followed by 25.5% (n = 13) with grade II procalcitonin, and 17.6% (n = 9) with grade III

procalcitonin.[11]

In our study, we found a statistically significant correlation between the serum procalcitonin level and the AIOS Score on days 1 and 2 as well as the CRP value.

According to a study by **Ratagiri et al.,** [11] children with severe pneumonia had higher levels of procalcitonin than children without the illness. **Don et al.**, [61] discovered that serum PCT was a helpful predictor of the severity of CAP in children when comparing the PCT values between those who required hospitalization and those who did not ($P < 0.0012$), as well as when comparing the interstitial pulmonary involvement and alveolar ($P < 0.0003$) on chest radiographs.

Serum PCT and CRP levels showed a positive linear connection ($r = 0.45$), according to a study by Yadav et al. 64. When the cut-off levels for differentiating between very severe and severe pneumonia were serum PCT levels >2 ng/ml and serum CRP levels >50 mg/dl, PCT's specificity was higher (86.4%) than CRP's (63.6%). The sensitivity was the same (92.9%). For serum PCT and CRP, the area under the receiver operating characteristics (ROC) curves was 0.923 and 0.837, respectively.

Compared to bronchopneumonia, **Lee et al.** found that both PCT and CRP were elevated in lobar pneumonia; however, only PCT was elevated in pneumonia with radiologic findings suggestive of severity.

Our research revealed a significant relationship between procalcitonin levels and outcome factors, while another study by **Instan et al.** found no differences in terms of morbidity or mortality. Our findings were found to conflict with these results.

STRENGTHS OF THE STUDY:

1. The research links a widely accepted clinical scoring method called AIOS to objective biomarker procalcitonin for better paediatric pneumonia diagnosis and severity evaluation.

2. The study provides early risk stratification capability by combining AIOS scoring with procalcitonin measurement to help detect high-risk patients so appropriate early interventions become possible and treatment results can be enhanced.

3. Disease progression kinetics become visible through the study's use of multiple day-to-day AIOS score checks which help validate treatment efficacy.

4. Results from the study demonstrate the meaningful relationship between high AIOS scores and elevated procalcitonin levels with patient death which provides crucial risk assessment capabilities.

5. Because AIOS functions as an easy bedside instrument it can serve clinical purposes in basic healthcare facilities including areas with limited resources.

6. The relationship between AIOS and the well-known bacterial infection biomarker procalcitonin enhances its clinical value for decision-making in medical contexts.

7. The study analyses actual hospital patient data from paediatric cases which enhances its practical value for standard clinical hospital work environments.

8. The research results can help promote antibiotic stewardship by showing which sick children need aggressive treatment while who can improve without prolonged antibiotic medications.

9. More extensive research will depend on this study to create standard procedures using AIOS and procalcitonin for assessing paediatric pneumonia severity.

10. Paediatric guidelines might receive input from this research to develop better early identification together with enhanced treatment approaches for paediatric pneumonia cases.

LIMITATIONS OF THE STUDY:

1. Generalization of research findings becomes difficult because the study took place in only one medical Centre.
2. A larger Sample size might strengthen the precision and accuracy of the study.

RECOMMENDATIONS:

1. Clinical staff should adopt regular AIOS utilization to detect early severity levels during paediatric pneumonia assessment, which helps guide treatment methods.

2. Procalcitonin functions as an important biomarker to supplement patient risk assessment and disease outcome prediction for community-acquired pneumonia (CAP) in children.

3. Regular AIOS scores taken daily between successive days help physicians gain enhanced understanding about how the disease advances and how treatments influence its course.

4. Future research needs to conduct bigger multi-centre tests which will validate the connection between AIOS and PCT while establishing definite threshold values useful for clinical decision support.

5. Healthcare staff should detect children with high AIOS scores and increased procalcitonin levels early for intense clinical attention along with ICU admission as required.

6. Evidence-guided antibiotic treatment allows healthcare providers to use procalcitonin levels for determining both start time and stop time of antibiotics, which helps prevent antibiotic overuse while decreasing antibiotic resistance.

7. Healthcare workers need training about AIOS scoring methods together with procalcitonin level interpretations in order to deliver faster medical diagnoses and treatment strategies.

8. AIOS scoring needs adjustment to operate in community healthcare facilities so severe pneumonia cases can begin their referral process to higher centers of care.

9. Due to present limitations, researchers should create and analyze predictive AI models that integrate AIOS values and procalcitonin measurements for automated assessment decisions.

10. Studies should follow children with high AIOS scores and elevations in procalcitonin through successive years to detect their subsequent diagnosis of infection recurrence alongside lung function deterioration and developmental progression.

CONCLUSION:

Both Acute Illness Observation Scale (AIOS) along with serum procalcitonin levels demonstrate strong ability to detect the severity level and determine clinical outcomes in paediatric patients with community-acquired pneumonia. Hence, we suggest that combining AIOS scoring system along with Serum Procalcitonin levels have a better clinical decision. The mortality risk increases when patients have high AIOS scores and their procalcitonin levels remain elevated. More extensive research using extensive participant numbers and extended observational periods is required to validate these results while developing standardized approaches for paediatric pneumonia care.

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ANNEXURE – I

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 968/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "STUDY OF ACUTE ILLNESS OBSERVATION SCALE AND SERUM PROCALCITONIN LEVELS IN COMMUNITY ACQUIRED PNEUMONIA IN CHILDREN AGED 2 MONTHS TO 59 MONTHS."

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.ANANYA PRAKASH

NAME OF THE GUIDE: DR. S.S.KALYANSHETTAR, PROFESSOR AND HOD, DEPT. OF PEDIATRICS.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Nalkwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303. Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE – II

RESEARCH INFORMED CONSENT FORM

BLDEA'S SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL &
RESEARCH CENTRE, VIJAYAPURA, KARNATAKA – 586103

TITLE OF THE PROJECT:

"**STUDY OF ACUTE ILLNESS OBSERVATION SCALE AND SERUM PROCALCITONIN LEVELS IN COMMUNITY-ACQUIRED PNEUMONIA IN CHILDREN AGED 2 MONTHS TO 59 MONTHS**"

GUIDE:
Dr. S. S. KALYANSHETTAR
PROFESSOR
DEPARTMENT OF PEDIATRICS

PG STUDENT:
Dr. ANANYA. PRAKASH

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to assess the "**STUDY OF ACUTE ILLNESS OBSERVATION SCALE AND SERUM PROCALCITONIN LEVELS IN COMMUNITY-ACQUIRED PNEUMONIA IN CHILDREN AGED 2 MONTHS TO 59 MONTHS**".

PROCEDURE:

I understand that after having obtained a detailed clinical history and thorough clinical examination and laboratory investigations, a final follow up of the serum magnesium levels and insulin resistance and its outcome is planned.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or my treatment. This is mainly the result of my condition and the procedure of this study are not expected to exaggerate these feelings, which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in this study will have no direct benefit to me other than the potential benefit of the research and education.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of the hospital record and will be subjected to confidentiality and privacy regulations. Information of a sensitive personal nature will not be part of medical records but will be stored in the investigations research file.

If the data is used for publication, the identity will not be revealed; other identifiers, such as photographs, will be used only with special permission. I understand that I may see the photograph before giving my permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study anytime. Dr. ANANYA. PRAKASH at the Department of Pediatrics will be available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the study that might influence my child's continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my child's participation is voluntary, and I may refuse to participate or withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. ANANYA. PRAKASH may terminate my participation in the study after she has explained the reasons

for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to my child resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to the child. However, no further compensation would be provided by the hospital. I understand that by my agreement to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

Dr. ANANYA. PRAKASH **Date:**

(Investigator)

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that Dr. ANANYA. PRAKASH is conducting a study on the "**STUDY OF ACUTE ILLNESS OBSERVATION SCALE AND SERUM PROCALCITONIN LEVELS IN COMMUNITY-ACQUIRED PNEUMONIA IN CHILDREN AGED 2 MONTHS TO 59 MONTHS**" In Shri B.M. Patel Medical College, Hospital & Research center, Vijayapura, Karnataka. Dr. ANANYA. PRAKASH has explained the purpose of the research and the study procedure. We are willing to give as much information as required for the study and consent for interventions and the possible discomforts and benefits. We have explained all the above in detail in our language, and we understand the same. Therefore, we agree to consent to our child's participation as a subject in this research project.

_____ _____

(Parents / Guardian) **(Witness to Signature)**

Date:

ANNEXURE – III

SCHEME OF CASE TAKING – PROFORMA

NAME:
IP NO:
AGE/SEX:
ADDRESS:
PH NO.:

HISTORY:

DURATION OF FEVER:
DURATION OF COUGH:
DURATION OF HURRIED BREATHING:
ASSOCIATED SYMPTOMS:
OTHER CO-MORBIDITIES:
PAST HISTORY:

CLINICAL EXAMINATION:

GENERAL PHYSICAL EXAMINATION:
HEART RATE:
RESPIRATORY RATE:
GRUNT:
TEMPERATURE:
CAPILLARY REFILL TIME:

SYSTEMIC EXAMINATION:

RS:
RETRACTIONS:
PERCUSSION:
BREATH SOUNDS:
ADDED SOUNDS:

CVS:
CNS:
P/A:

NEED OF OXYGEN: YES/NO
PRIMARY MODE OF RESPIRATORY SUPPORT:
FIRST LINE ANTIBIOTICS:
SECOND LINE ANTIBIOTICS, IF UPGRADED:
SURGICAL PROCEDURES, IF ANY:

AIOS SCORING:

OBSERVATION	1 (NORMAL)	3(MODERATE IMPAIRMENT)	5(SEVERE IMPAIRMENT)
QUALITY OF CRY	Strong with normal tone or content with no cry	Whimpering or sobbing	Weak or moaning or high pitched
REACTION TO PARENT STIMULATION	Cries briefly and stops or content with no cry	Cries off and on	Continual cry or hardly responds
STATE VARIATION	If awake, stays awake. If sleeping and stimulated, wakes up quickly	Eyes close briefly awake or awakes with prolonged stimulation	Falls to sleep or will not arouse
COLOR	Pink	Pale extremities or acrocyanosis	Pale or cyanotic or mottled
HYDRATION	Skin and eyes normal and mucus membrane moist	Skin and eyes normal and mouth slightly dry	Skin doughy/tented and dry mucus membrane and/or sunken eyes
RESPONSE TO SOCIAL OVERTURES	Smiles	Brief smile or alerts briefly	No smile, anxious face, dull, expressionless or no alerting

TOTAL SCORE: OUT OF 30
GRADING OF AIOS SCORE: MILD/MODERATE/SEVERE
(<11-Mild, 11-15-Moderate, >15-Severe)
AT DAY 1:
GRADE:
AT DAY 2:
GRADE:
AT DAY 5:
GRADE:

INVESTIGATIONS:

	ON ADMISSION	IF REPEATED
Total Count		
Hemoglobin		
Platelet count		
CRP Grade I: <10mg/L (Normal) Grade II: 10 – 25mg/L (Mild) Grade III: 26 -50mg/L (Moderate) Grade IV: >50.0mg/L (Severe)		
Procalcitonin Grading: Normal/Mild/Moderate/Severe <0.1ug/L- Normal 0.1-0.25ug/L- Mild 0.25-0.5ug/L- Moderate >0.5ug/L- Severe		
Blood Culture		

DURATION OF STAY IN HOSPITAL:
DEATH/DISCHARGE:

ANNEXURE – IV

BIODATA OF GUIDE

Name: DR. S.S KALYANSHETTAR

Date of Birth: 17/1/1974

Education: M.B.B.S, MD

Present Designation: Professor, Dept of

Pediatrics, B.L.D.E (Deemed to be University),

Shri. B.M. Patil Medical College, Vijayapura,

Karnataka.

Registration No: 45576

Work experience: 18 years

Membership: Indian Academy of Pediatrics

BIODATA OF POST-GRADUATE

Name: DR ANANYA PRAKASH

Date of Birth: 08/02/1998

Age: 27 years

Qualification: MBBS

Registration No.: 143380

Designation: Post Graduate Student

Department of Paediatrics

Address: New PG Hostel, Shri B M Patil Medical College Hospital, Vijayapura, Karnataka 586103

ANNEXURE – V

MASTER CHART

	I	IP	Age	A	S	D	Cough	I	L			Pas	Ge	Hea	R	I	Te	C	F	Pe	Air			Card	I		Nee
##	Sar	####	1	1 yea	Fem	2	1	1	1	Nil	I	Similar comp		98	44	Pre	104	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	An	####	1	6 mo	Fem	10	2	10	Nil	Nil	I	Simila	Pallor	100	54	Ab	100	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Na	####	1	3 mo	Fem	2	2	15	1	Vo	I	Nil	Pallor	140	68	Pre	104	=3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Irrit	So	Yes
##	Ve	####	1	5 mo	Mal	2	1	4	1	Nil	S	Similar comp		120	52	Ab	100	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Shi	####	3	5 ye	Fem	3	1	3	1	Nil	S	Nil		98	46	Ab	100	<3 s	Sub	Bilate	Bilate	Bilater		S1, S2	Cor	So	Yes
##	Nir	####	2	2 ye	Mal	4	1	4	2	Nil	S	Nil	Pallor	130	50	Ab	102	<3 s	Sub	Stony	Bilate	Bilater		S1, S2	Cor	So	Yes
##	Ga	####	1	5 mo	Mal	1 \		Nil	1	Nil	I	Nil	Pallor	110	50	Ab	101	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ary	####	3	5 ye	Mal	5	3	Nil	1	Nil	I	Nil		100	44	Ab	102	<3 s	Sub	Dull r	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Su	####	3	4 ye	Mal	1	1	3	3	Nil	I	Repeated epi		122	60	Ab	102	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Sh	####	2	3 ye	Fem	3	1	2	Nil	Nil	I	Similar comp		98	42	Ab	103	<3 sec		Bilate	Bilate	Left si		S1, S2	Cor	So	Yes
##	Har	15675	1	1 yea	Mal	3	1	2	2	Nil	I	Similar comp		106	40	Ab	99	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Am	####	2	3 ye	Fem	4	1	4	1	Nil	I	Simila	Edem	112	60	Ab	100	<3 s	Sub	Dullr	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Sh	####	1	1 yea	Fem	3	3	Nil	1	Vo	I	Nil	Pallor	132	62	Pre	99	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Pra	####	2	2 ye	Mal	4	1	4	2	Lo	(	Simila	Pallor	150	65	Pre	102	=3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Adi	####	3	4 ye	Fem	2	1	2	Nil	Nil	I	Nil		94	24	Ab	100	<3 sec		Bilate	Bilate	Bilater		S1, S2	Cor	So	Yes
##	Var	####	3	4 ye	Fem	7	1	7	2	Nil	I	Nil		114	36	Ab	99	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Joh	####	1	1 yea	Mal	5	1	3	1	Nil	I	Admis	Pallor	100	24	Ab	98	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Shi	####	1	1 yea	Fem	5	1	5	Nil	Nil	I	Similar comp		98	30	Ab	97	<3 sec		Bilate	Bilate	Bilater		S1, S2	Cor	So	No
##	Ray	####	1	1 yea	Mal	3	1	3	1	Vo	I	Simila	Pallor	120	34	Ab	99	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ary	####	3	4 ye	Fem	4	1	3	1	nil	I	Nil	Pallor	100	34	Pre	100	>3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ad	####	1	1 yea	Mal	3	1	2	1	Po	I	Simila	Pallor	120	48	Pre	100	=3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ad	####	2	1 yea	Mal	3	1	2	1	Po	I	Simila	Pallor	120	48	Pre	100	=3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	hear	Co	Yes
##	Ma	9524	1	19 m	Mal	1	3	Nil	1	Nil	S	Nil	Pallor	150	38	Ab	99	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Abl	527	2	2 ye	Mal	7	1	7	2	Ab	I	Nil	Pallor	180	70	Pre	103	>3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Sp	####	1	11 m	Fem	3	1	2	1	Nil	I	Surge	Pallor	150	50	Ab	101	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Nik	1452	2	3 ye	Mal	2	1	2	2	Nil	I	Nil	Pallor	122	36	Ab	101	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ad	1124	2	3 ye	Fem	2	3	Nil	1	Me	'	Nil	Pallor	128	24	Ab	101	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Adi	1893	1	5 mo	Mal	1	3	Nil	Nil	Co	(	Similar comp		122	28	Ab	100	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Abl	2880	1	1 yea	Mal	4	1	4	1	Nil	I	Simila	Pallor	128	62	Ab	101	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Zur	####	1	8 mo	Fem	5	1	2	1	Co	S	Nil	Pallor	118	36	Pre	101	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Vis	3516	2	3 ye	Mal	4	1	4	1	Nil	I	Viral I	Pallor	132	40	Ab	103	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Blo	3675	1	2 mo	Fem	3	3	Nil	2	Nil	I	Nil		150	66	Ab	101	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Sar	3517	2	3 ye	Mal	4	1	4	1	Nil	I	Similar comp		122	46	Ab	101	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Vir	2487	1	3 mo	Mal	2	3	Nil	1	Vo	I	Nil		110	50	Ab	101	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Aka	3715	2	3 ye	Mal	2	1	4	1	Co	I	Nil	Pallor	140	58	Ab	101	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Sa	4273	1	10 m	Fem	2	3	Nil	2	Nil	F	Previo	Cyano	160	70	Pre	102	=3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ve	3708	1	18 m	Mal	1	1	4	Nil	Nil	I	Nil		160	60	Ab	100	<3 sec		Bilate	Bilate	Bilater		S1, S2	Cor	So	Yes
##	Sor	5121	1	7 mo	Mal	2	1	7	Nil	Nil	F	Nil		130	62	Ab	102	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Be	5523	3	5 ye	Mal	3	1	1	1	Vo	I	Nil		128	40	Ab	101	<3 sec		Dull r	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ath	4974	1	6 mo	Mal	5	1	2	1	Nil	I	Nil	Pallor	130	52	Pre	103	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Nag	5518	3	5 ye	Mal	3	1	3	1	Nil	F	Nil		120	50	Pre	102	<3 s	Sub	Dull r	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ra	5355	1	8 mo	Mal	1	1	3	1	Nil	I	Nil	Pallor	126	28	Ab	101	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Ku	4738	3	4 ye	Mal	2	1	4	Nil	Pa	I	Similar comp		110	40	Ab	102	<3 s	Sub	Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Shi	5155	1	6 mo	Mal	3	1	3	1	Nil	I	Nil	Pallor	102	32	Ab	101	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes
##	Par	6278	2	3 ye	Mal	3	1	3	2	Ch	I	Nil		138	50	Ab	102	<3 sec		Bilate	Bilate	Bil	Bila	S1, S2	Cor	So	Yes

I	Prin	I	S	S	AIDS	Gra	A	Gr	All	G				H	Plate	CR	G	Se	G	Bl	D	Out
Sar	Nasal F	Piperaci		Nil	14	Moder	12	Mode	8	Mild	##	73	#	12	6E+05	9.8	II	0.01	I	Steril	5	Improved and Discharged
An	Nasal F	Pip	Ose	Nil	6	Mild	6	Mild	6	Mild	##	59	#	11	2E+05	<5	I	0.02	I	Steril	15	Improved and Discharged
Na	Conver	Pip	Men	Nil	26	Severe	28	Seve	28	Seve	##	33	#	12	5E+05	36.8	III	99.3	IV	Steril	17	Death
Ve	HFNC	Piperaci		Nil	8	Mild	8	Mild	6	Mild	##	25	#	11	4E+05	<5	I	0.48	I	Steril	6	Improved and Discharged
Shi	Conver	Pip	Men	Nil	26	Severe	28	Severe			##	36	#	11	1E+05	14.8	III	115	IV	Steril	4	Death
Nir	HFNC	Meroper		Inte	14	Moder	12	Mode	8	Mild	##	64	#	7.3	3E+05	>90	IV	38.8	IV	Steril	19	Improved and Discharged
Ga	HFNC	Cef	Line	Nil	14	Moder	10	Mild	6	Mild	##	52	#	12	4E+05	<5	I	0.15	I	E coli	7	Improved and Discharged
Ary	HFNC	Pip	Van	Inte	18	Severe	14	Mode	10	Mild	##	94	5	11	8E+05	74	IV	50.6	IV	Steril	17	Improved and Discharged
Su	Conver	Pip	Van	Nil	14	Moder	10	Mild	6	Mild	##	36	#	11	3E+05	7	II	11.8	IV	Steril	10	Improved and Discharged
Sh	HFNC	Pip	Ose	Nil	14	Moder	12	Mode	8	Mild	##	64	#	12	2E+05	43	III	17.9	IV	Steril	7	Improved and Discharged
Har	Nasal F	Amoxyc		Nil	8	Mild	6	Mild	6	Mild	##	25	#	11	4E+05	<5.0	I	0.2	I	Steril	7	Improved and Discharged
Am	HFNC	Me	Ose	Nil	13	Moder	13	Mode	8	Mild	##	60	#	11	2E+05	121	IV	29.1	III	Steril	6	Improved and Discharged
Sh	HFNC	Piperaci		Nil	14	Moder	10	Mild	8	Mild	##	82	#	6	2E+05	83	IV	46.2	IV	Steril	6	Improved and Discharged
Pra	HFNC	Pip	Men	Nil	15	Moder	27	Severe			##	50	#	8.1	3E+05	44.7	III	100	IV	Steril	2	Death
Adi	Nasal F	Cefotaxi		Nil	10	Mild	10	Mild	6	Mild	##	48	#	10	1E+05	47.9	III	0.67	II	Steril	5	Improved and Discharged
Var	Nasal F	Amikaci		Nil	8	Mild	6	Mild			##	60	#	12	2E+05	34.3	III	1.25	II	Steril	3	Improved and Discharged
Joh	HFNC	Piperaci		Nil	14	Moder	10	Mild	6	Mild	##	82	#	12	5E+05	0.4	I	0.33	I	Steril	7	Improved and Discharged
Shi		Am	Ose	Nil	6	Mild	6	Mild	6	Mild	##	40	#	11	2E+05	5.2	I	0.2	I	Steril	3	Improved and Discharged
Ray	HFNC	Amoxyc		Nil	10	Mild	8	Mild	6	Mild	##	63	#	9	3E+05	16.3	II	0.95	II	Steril	5	Improved and Discharged
Ary	Conver	Pip	Men	Nil	26	Severe	28	Severe			##	49	#	11	69000	42.8	III	68.1	IV	Staph	3	Death
Ad	HFNC	Pip	Ose	Nil	14	Moder	26	Severe			##	51	#	9	5E+05	28	III	100	IV	Coag	4	Improved and Discharged
Ad	HFNC	Pip	Ose	Nil	14	Moder	26	Severe			##	51	#	9	5E+05	9	II	100	IV	Coag	4	Death
Ma	HFNC	Pip	Ose	Nil	14	Moder	10	Mild	6	Mild	##	33	#	9.3	2E+05	<5	I	0.79	II	Steril	7	Improved and Discharged
Abl	Conver	Piperaci		Nil	26	Severe					##	71	#	8.6	97000	58.6	IV	18.8	IV	Steril	1	Death
Sp	Nasal F	Piperaci		Nil	10	Mild	8	Mild			##	66	#	8.9	5E+05	38	III	3.32	III	Steril	4	Improved and Discharged
Nik	Nasal F	Am	Ose	Nil	8	Mild	6	Mild			##	66	#	9.8	4E+05	13.9	II	1.57	II	Steril	3	Improved and Discharged
Ad	Nasal F	Me	Metr	Nil	8	Mild	6	Mild	6	Mild	##	42	#	2.9	91000	<5	I	0.07	I	Steril	7	Improved and Discharged
Adi	Nasal F	Ceftriax		Nil	8	Mild	8	Mode	6	Mild	##	42	#	10	7E+05	<5	I	0.05	I	Steril	5	Improved and Discharged
Abl	HFNC	Pip	Ose	Nil	16	Severe	26	Severe			##	48	#	9.8	2E+05	10.2	III	0.07	I	Steril	2	Death
Zur	HFNC	Piperaci		Nil	12	Moder	10	Mild	6	Mild	##	44	#	12	3E+05	13.7	III	0.52	II	Steril	6	Improved and Discharged
Vis	Nasal F	Pip	Ose	Nil	8	Mild	8	Mild	6	Mild	##	26	#	6.5	3E+05	8	II	47.7	IV	Steril	10	Improved and Discharged
Blo	HFNC	Piperaci		Nil	14	Moder	10	Mild	6	Mild	##	11	#	10	6E+05	13.5	III	0.65	II	Steril	8	Improved and Discharged
Sar	HFNC	Am	Van	Nil	14	Moder	10	Mild	6	Mild	##	82	#	8	7E+05	>90	IV	6.87	III	Steril	12	Improved and Discharged
Vir	Nasal F	Piperaci		Nil	10	Mild	8	Mild	6	Mild	##	52	#	9	4E+05	10	II	0.52	II	Steril	8	Improved and Discharged
Aka	HFNC	Piperaci		Nil	18	Severe	12	Mode	8	Mild	##	68	#	10	6E+05	<5	I	0.17	I	Steril	10	Improved and Discharged
Sa	Conver	Pip	Ose	Nil	28	Severe	18	Seve	12	Mod	##	64	#	11	2E+05	13.3	III	1.11	II	Steril	14	Improved and Discharged
Ve	Nasal F	Pip	Ose	Nil	12	Moder	10	Mild	8	Mild	##	68	#	8.4	6E+05	>90	IV	2.23	III	Steril	8	Improved and Discharged
Sor	Nasal F	Piperaci		Nil	12	Moder	10	Mild	6	Mild	##	15	#	8.6	3E+05	>90	IV	2.66	III	Steril	7	Improved and Discharged
Be	HFNC	Pip	Ose	Bila	12	Moder	16	Seve	8	Mild	##	85	#	6.5	3E+05	535	IV	51.8	IV	Steril	18	Improved and Discharged
Ath	HFNC	Pip	Azitl	Nil	14	Moder	10	Mild	6	Mild	##	39	#	9.4	6E+05	>90	IV	8.83	III	Steril	6	Improved and Discharged
Nag	HFNC	Pip	Ose	Tho	12	Moder	22	Seve	14	Mod	##	92	5	12	3E+05	>90	IV	8.23	III	Steril	35	Improved and Discharged
Ra	Nasal F	Piperaci		Nil	12	Moder	10	Mild	6	Mild	##	39	#	8.8	5E+05	14.6	III	1.9	II	Steril	5	Improved and Discharged
Ku	HFNC	Pip	Ose	Left	12	Moder	10	Mild	10	Mild	##	65	#	10	55000	>90	IV	0.55	II	Steril	10	Improved and Discharged
Shi	HFNC	Pip	Ose	Nil	14	Moder	8	Mild	6	Mild	##	4	#	9.3	3E+05	75.6	IV	90.9	IV	Steril	9	Improved and Discharged
Par	HFNC	Pip	Ose	Nil	12	Moder	8	Mild	8	Mild	##	78	#	11	4E+05	>90	IV	3.3	III	Steril	10	Improved and Discharged

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