"PREVALENCE OF "VITAMIN A" DEFICIENCY IN CHILDREN AGED 2MONTHS TO 5 YEARS WITH PNEUMONIA; A HOSPITAL BASED, CROSS SECTIONAL STUDY"

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ABBREVIATIONS

1. ARI – Acute Respiratory Infection

2. BTS – British Thoracic Society

3. CAP – Community-Acquired Pneumonia

4. COVID-19 – Coronavirus Disease 2019

5. CRP – C-reactive Protein

6. CT – Computed Tomography

7. CTRI – Clinical Trial Registry of India

8. CXR – Chest X-ray

9. DEVTA – Deworming and Vitamin A Trial

10. ELISA – Enzyme-Linked Immunosorbent Assay

11. GAPP – Global Action Plan for Prevention and Control of Pneumonia

12. HIV – Human Immunodeficiency Virus

13. HRP – Horseradish Peroxidase

14. ICU – Intensive Care Unit

15. IgA – Immunoglobulin A

16. IQ – Interquartile Range

17. JAMA – Journal of the American Medical Association

18. KAHER – KLE Academy of Higher Education and Research

19. KLEH – KLE Hospital

20. LMIC – Low- and Middle-Income Countries

21. LRTI – Lower Respiratory Tract Infection

22. MRI – Magnetic Resonance Imaging

23. mL – Milliliters

24. N Engl J Med – The New England Journal of Medicine

25. NIS – National Immunization Schedule

26. OR – Odds Ratio

27. p – Probability Value

28. q – Complementary Probability (100 - p)

29. RA – Retinoic Acid

30. RBP – Retinol Binding Protein

31. RE – Retinyl Esters

32. RPM – Revolutions Per Minute

33. RR – Respiratory Rate

34. SARS-CoV-2 – Severe Acute Respiratory Syndrome Coronavirus 2

35. SD – Standard Deviation

36. UI – Uncertainty Interval

37. VA – Vitamin A

38. VAD – Vitamin A Deficiency

39. VADD – Vitamin A Deficiency Disorders

40. VMNIS – Vitamin and Mineral Nutrition Information System

41. WHO – World Health Organization

42. Z – Standard Normal Variable Value

ABSTRACT

PREVALENCE OF "VITAMIN A" DEFICIENCY IN CHILDREN AGED 2MONTHS TO 5 YEARS WITH PNEUMONIA ; A HOSPITAL BASED, CROSS SECTIONAL STUDY

Background: Vitamin A deficiency (V AD) is a significant public health concern, particularly among children with pneumonia, affecting immune function and disease severity. Limited data exist on the prevalence and impact of VAD in North Karnataka. This study aimed to assess the burden of VAD in children with pneumonia and its association with disease severity, ICU stay, and outcomes.

Methods: A cross-sectional observational study was conducted among children diagnosed with pneumonia. Baseline demographic and clinical data, including vitamin A levels, pneumonia severity, duration of ICU stay, and treatment outcomes, were collected. The prevalence of subclinical and severe VAD was determined, and statistical analysis was performed to assess correlations between vitamin A status and pneumonia severity.

Results: Among the study population, 42.7% had subclinical VAD, while 27.1% had severe deficiency. Severe VAD was strongly associated with an increased risk of severe pneumonia, higher need for invasive ventilation, and a mortality rate of 42.3%. Children from rural areas exhibited significantly higher rates of VAD and severe pneumonia compared to urban children. Although the overall recovery rate was 82.2%, children with severe VAD had a longer ICU stay and worse clinical outcomes.

Conclusion: This study highlights the high prevalence of VAD in children with pneumonia and its substantial impact on disease severity and outcomes. Strengthening vitamin A supplementation programs, improving nutritional education, and implementing targeted public health interventions could help mitigate the burden of VAD and improve child health outcomes.

Keywords: Vitamin A deficiency, pneumonia, child health, immune function, North Karnataka

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INTRODUCTION

Pneumonia is an acute infection of the lung parenchyma that leads to inflammation and alveolar filling with fluid or pus, causing impaired gas exchange.. Viruses, bacteria, and other pathogens are all potential causes of this condition. Pneumonia remains the leading cause of death among children all across the world, with India being responsible for twenty percent of these deaths and having a higher burden of childhood pneumonia than any other country.[1] At the national level, the estimated number of pneumonia cases among children who were not infected with HIV in India had a significant decrease from 83·8 million cases in the year 2000 to 49·8 million cases in the year 2015, with the standard uncertainty interval (UI) ranging from 14·0 to 300·8. State-level estimates provide light on the significant disparity that exists between states in terms of the incidence of the problem and the work that has been made in mitigating it. More than half of all children under the age of five were predicted to have pneumonia in the states of Uttar Pradesh and Madhya Pradesh in the year 2015. In Uttar Pradesh, there were 565 cases per 1000 children [95% UI 94–2047], while in Madhya Pradesh, there were 563 cases per 1000 children [88–2084]. In contrast, the states of Kerala and Tamil Nadu had the lowest prevalence of pneumonia, with 137 cases per 1000 children in Kerala and 169 cases per 1000 children in Tamil Nadu respectively.[2] Despite the fact that there is still a significant amount of work to be done across the entirety of India, the decreases in the number of cases of pneumonia that were recorded in Kerala and Tamil Nadu between the years 2000 and 2015 can provide valuable insights for developing strategies that can lead to equivalent reductions in other states and nations.[3]

Over 700,000 children under the age of five lose their lives to pneumonia every year, which is equivalent to almost 2,000 deaths per day. Pneumonia is the

infectious disease that causes the most deaths among children. More than 190,000 infants are included in this total. The vast majority of these fatalities might have been avoided. Every year, there are approximately 1,400 cases of pneumonia per 100,000 children, which is equivalent to one case per 71 children. The highest prevalence of pneumonia is seen in South Asia, where there are 2,500 cases per 100,000 children, and in West and Central Africa, where there are 1,620 cases per 100,000 children. Compared to other infectious diseases, the rate of progress in reducing the number of deaths that are caused by pneumonia in children under the age of five has been substantially slower. Pneumonia-related mortality among children under the age of five have fallen by 54% since the year 2000, whereas deaths caused by diarrhoea have decreased by 63% during the same time period.[4]

The vast majority of these deaths, which are likely to have been preventable, take place in settings with limited resources and are strongly connected to poverty, inadequate access to health care, and undernutrition.[5] It is generally known that a lack of vitamin D can lead to recurrent respiratory tract infections, and as a result, the practice of supplementing with vitamin D has been integrated into clinical practice.[6] Similarly, it has been discovered that vitamin E and zinc both play a preventive function against respiratory and gastrointestinal illnesses; nevertheless, the practice of routine supplementation is not being carried out on a regular basis.[7]

On the other hand, another micronutrient that is as important is vitamin A, whose connection to lower respiratory tract infections (LRTIs) like pneumonia has not been thoroughly investigated and documented. Deficiency in vitamin A is another issue that affects public health in many regions of the world, particularly in Africa and South-East Asia. A lack of vitamin A can lead to night blindness, which is a sort of visual impairment. Additionally, a lack of vitamin A may increase the likelihood of

being unwell or dying from childhood diseases, such as measles and those that cause diarrhoea. It has been established through scientific trials that taking vitamin A supplements can lessen the severity of respiratory infections and mortality rates in children who have measles. Vitamin A is a crucial component in the fight against illnesses that affect toddlers and children. On the other hand, the findings of studies conducted on children who did not have measles indicate that vitamin A does not have a significant protective impact. [8]

There are a variety of nutrition interventions that have been demonstrated to be beneficial in lowering the number of cases of acute lower respiratory tract infections and the potentially fatal outcomes that are linked with pneumonia. Vitamin A supplementation has been examined as a potential intervention to expedite recovery, reduce the severity of acute lower respiratory tract infections, and prevent recurrent episodes of these illnesses[9-14]. This is due to the fact that vitamin A has been shown to be beneficial in guarding against measles-associated pneumonia[15]. In no way have the results been consistent with one another. Some writers have claimed that there are no advantages[16-19], while others have only indicated good effects for particular groups, such as children who are underweight[20] or those who have a pre-existing vitamin A deficit[21]. Additionally, it has been discovered that using vitamin A supplements can lead to an increase in the occurrence of acute lower respiratory tract infections, particularly in children who have higher food intakes[20,22].

It would appear that children who are deficient in vitamin A are at a larger risk of being ill and experiencing death as a result of respiratory tract infections[23]. It has been demonstrated that taking vitamin A supplements can reduce the risk of death in children aged 6–59 months by around 23–30%[24]. Foods that contain a considerable amount of vitamin A, such as animal products (liver, milk, cheese, eggs), or foods that

have been fortified with vitamin A may not be consumed regularly in environments that have a limited supply of resources and a high incidence of acute lower respiratory tract infections[25]. Under these conditions, it may therefore be required to improve availability to foods that are high in provitamin A, such as mangoes and papayas, through the implementation of dietary diversity and household food production programs[26-28].

Nevertheless, there are not many research that provide evidence that a lack of vitamin A is a micronutrient that contributes to the development of pneumonia. Due to the fact that supplementation programs are already in existence, it has been discovered through an examination of representative survey data that two out of every five children in India who are eligible for the program are not receiving preventive vitamin A supplementation[8]. This deprives a significant portion of the population of the opportunity to reap the benefits of this program. It is possible for LRTIs to affect this particular segment of the population. It is possible that the typical practice of administering antibiotics to treat the RTI may just treat the symptoms of the infection on an occasional basis, without treating the underlying cause that caused the infection to repeat so frequently. The symptoms of deficiency do not appear to be displayed by a significant section of the population, and as a result, they are not detected. This phenomenon occurs even in industrialised countries. These findings are particularly pertinent in developing nations such as India, where there have been relatively few studies conducted in this particular area. The primary objectives of this research are to determine the prevalence of Vitamin A deficiency (VAD) in children who have pneumonia and to assess the correlation between serum vitamin A levels and the severity of pneumonia, with a special focus on subclinical VAD. If this association between the latent deficiency in micronutrients and the patent symptoms of

pneumonia is established, it will not only stimulate additional study but also provide a boost for the vitamin A supplementation program, which will go a long way towards reducing mortality. Moreover, this will guarantee an improvement in the health conditions of children and a large reduction in the amount of school absenteeism that they experienced.

AIMS AND OBJECTIVES

- To study the prevalence of Vitamin A deficiency in children with Pneumonia.
- To study the association between Vitamin A Levels and severity of Pneumonia and its outcome.

REVIEW OF LITERATURE

PNEUMONIA:

Pneumonia is an infection of the lower respiratory tract that is characterised by inflammation of the lung parenchyma[29]. Children and adolescents are most likely to get infected with respiratory tract infections (RTIs), which are the most common infectious disease. The high morbidity and mortality rates in developing countries make this one among the most important health problems in these countries. It is responsible for more than 9 lakh fatalities per year and is responsible for 14% of infant mortality worldwide, and it accounts for roughly 18% of infant mortality in India. [30,31]

There is a significant correlation between childhood pneumonia and morbidity and death in nations with extensive resources, as well as morbidity and mortality in countries with inadequate resources. Lower respiratory tract infections (LRTIs) were responsible for over 800,000 deaths among children aged 19 and under across the globe in the year 2015, with a rate of 31.1 deaths per 100,000 population. This represents the second highest rate, after neonatal/preterm birth complications[32]. Based on observational studies conducted in nations with sufficient resources, the rate of fatalities among hospitalised children under the age of five was shown to be less than one percent[33,34]. During a comprehensive analysis, it was shown that the case fatality rate among hospitalised children under the age of five in countries with low resources varied from 0.3 to 15 percent[33].

Risk factors — Lower socioeconomic classes have a higher prevalence of lower respiratory tract infections (LRTIs), which correlates well with family size, which is a reflection of urbanisation in the environment. It is common for children of school age

to bring respiratory viral agents into their homes, which can lead to secondary illnesses in their carers and siblings[35]. The presence of underlying cardiovascular problems and other medical illnesses makes a person more susceptible to developing pneumonia and contributes to the severity of the condition. Examples of this include:[36,37] Some of the conditions that are considered to be neuromuscular diseases include congenital heart disease, bronchopulmonary dysplasia, cystic fibrosis, asthma, sickle cell disease, and neuromuscular disorders, particularly those that are accompanied with a lowered consciousness. Particular gastrointestinal conditions, such as gastro-oesophageal reflux and tracheo-oesophageal fistula, as well as congenital and acquired immunodeficiency disorders, are included in this category.

PATHOGENESIS

Pneumonia can be caused by a number of factors, including the infiltration of a virulent organism, the infiltration of an overwhelming inoculum, or the damage of the host's defences. In the typical situation, pneumonia is the result of an illness that affects the upper respiratory tract and allows bacteria, viruses, or other pathogens to invade the lower respiratory tract. This invasion causes the immune system to react and causes inflammation[38,39]. It is common for white blood cells, fluid, and debris from cells to accumulate in the air gaps of the lower respiratory tract. As a consequence of this process, lung compliance is decreased, resistance is increased, smaller airways are obstructed, and the collapse of distal air gaps, air trapping, and altered ventilation-perfusion relationships may occur[38]. There is a correlation between severe infection and necrosis of the bronchial or bronchiolar epithelium[40] and/or pulmonary parenchyma[41].

Acquisition — It is most common for the agents that cause infections of the lower respiratory tract to be transferred through droplet dissemination, which occurs when

an individual comes into close contact with a source case. Additionally, it is possible that contact with contaminated fomites plays a significant role in the acquisition of viral agents, particularly respiratory syncytial virus. The majority of common bacterial pneumonias, such as S. pneumoniae, are caused by the initial colonization of the nasopharynx, which is then followed by the aspiration or inhalation of organisms.

Normal host defense — Anatomic and mechanical barriers, humoral immunity, phagocytic activity, and cell-mediated immunity are all components of the pulmonary host defense system, which is a complicated system[42,43].

Anatomic and mechanical barriers – There are both anatomical and mechanical barriers in the upper airway, and they are an essential component of the host's defence mechanism. It is possible for particles that are larger than 10 microns to be effectively filtered out by the hairs in the anterior nares or to become caught on the mucosal surfaces. The nasal mucosa is composed of mucus-producing cells as well as ciliated epithelial structures. The cilia pulse in unison, releasing the organisms that have become trapped through the nasopharynx through either swallowing or ejection. The flow of saliva, the sloughing of epithelial cells, the local synthesis of complement and immunoglobulin (Ig)A, and the interference of bacteria from the resident flora are all essential elements in the local host defence that occurs in the oropharynx.

Both the cough reflex and an intact epiglottic reflex have a role in preventing the aspiration of contaminated fluids. The cough reflex also plays a role in the movement of materials that could be aspirated. When particles measuring between 5 and 10 microns collide with mucosal surfaces, they become imprisoned in endobronchial mucus. This is because the steep angles at which the central airways branch lead the particles to strike. After being ensnared, the particles are moved

upward by the ciliary system, which then takes them out of the airways and into the throat, where they are often swallowed.

Humoral immunity – The most important immunoglobulin that is produced in the upper airways is called secretory IgA, and it is responsible for ten percent of the total protein content that is found in nasal secretions. In spite of the fact that it is not a particularly effective opsonising agent, it does have antibacterial and antiviral action. The majority of the time, IgG and IgM are transported from the bloodstream into the alveolar spaces and airways. Once there, they perform the functions of opsonising bacteria, activating complement, and neutralizing toxins. Complement, immunoglobulins, surfactant, and fibronectin are all examples of efficient opsonins that can assist in the elimination of microorganisms (particles ranging from 0.5 to 1 micron) that have reached the terminal airways and alveoli within the body. There is also the presence of iron-binding proteins, free fatty acids, and lysozyme, all of which have the potential to kill microbes.

Phagocytic cells – In the lung, there are two different groups of phagocytic cells: macrophages and polymorphonuclear leukocytes that originate from the blood. Macrophages can be classified into a number of unique populations, each of which differs in both their location and their function.

It is the first phagocyte that inert particles and potential pathogens that enter the lung come into contact with. The alveolar macrophage is found in the alveolar fluid and is the first encountered phagocyte. This cell has the potential to become a mediator of inflammation and to create cytokines that recruit neutrophils if it is subjected to an inadequate amount of stimulation. These cells, known as interstitial macrophages, are found in the connective tissue of the lungs and perform the functions of both phagocytic cells and antigen-processing cells.

These macrophages are found in the capillary endothelial cells of the intravascular space. They are responsible for phagocytosing and removing foreign material that has entered the lungs through the bloodstream.

Cell-mediated immunity – When it comes to specific infections, such as viruses and intracellular bacteria that are able to live within pulmonary macrophages, cell-mediated immunity is particularly crucial. Despite the fact that lymphocytes make up just five to ten percent of the overall population of lung parenchyma cells, they are responsible for three extremely important functions: the generation of antibodies, the production of cytokines, and the development of cytotoxic activity.

Pathologic patterns of pneumonia — There are five pathologic patterns of bacterial pneumonia[39]:

Lobar pneumonia – Participation of a single lobe or a portion of a lobe in the process. This is the typical pattern of pneumonia caused by S. pneumoniae organisms.

- Bronchopneumonia – Airways and the interstitium that surrounds them are the primary sites of interaction. There are instances in which this pattern is observed in pneumonia caused by Streptococcus pyogenes and Staphylococcus aureus.
- Necrotizing pneumonia (associated with aspiration pneumonia and pneumonia resulting from S. pneumoniae, S. pyogenes, and S. aureus).
- Caseating granuloma (as in pneumonia caused by Mycobacterium tuberculosis and the endemic mycoses).
- Interstitial and peribronchiolar with subsequent parenchymal infiltration is a pattern that often manifests itself when a severe case of viral pneumonia is worsened by bacterial pneumonia.

There are two major pathologic patterns of viral pneumonia[39]:

Interstitial pneumonia , Parenchymal infection

ETIOLOGIC AGENTS

Common etiologic agents of pediatric pneumonia*

MICROBIAL AGENT	SUSCEPTIBLE HOSTS
Bacteria	
Chlamydia trachomatis	First 3 months after birth
Mycoplasma hominis	First 3 months after birth
Treponema pallidum	First 3 months after birth
Ureaplasma urealyticum	First 3 months after birth
Staphylococcus aureus	Primarily children <5 years
Streptococcus pyogenes	Primarily children <5 years
Chlamydia pneumoniae	Primarily children ≥5 years
Mycoplasma pneumoniae	Primarily children ≥5 years
Streptococcus pneumoniae	All
Viruses	
Respiratory syncytial virus	Primarily children <5 years
Influenza A and B	Primarily children <5 years
Human metapneumovirus	Primarily children <5 years
Parainfluenza viruses 1, 2, and 3	Primarily children <5 years
Coronaviruses (229E C43, NL63, HKU1)	Primarily children <5 years
Adenoviruses	Primarily children <5 years
Rhinovirus	Primarily children <5 years

CLINICAL FEATURES

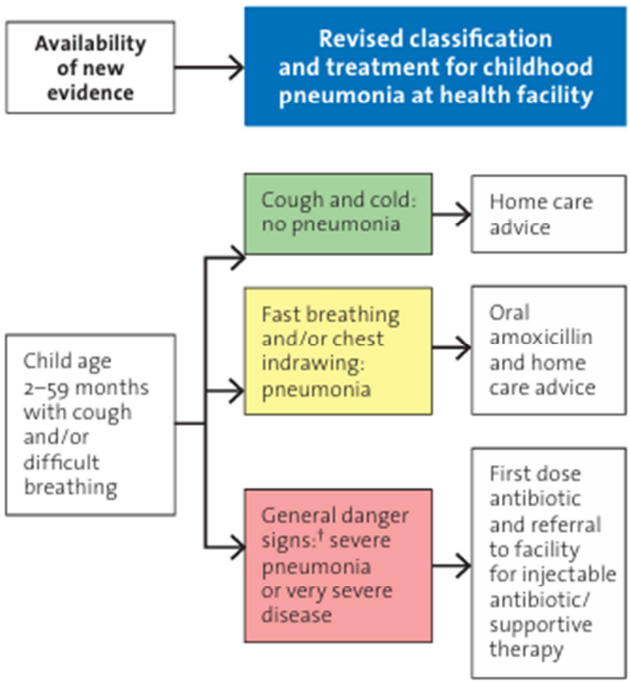
Symptoms

When children are diagnosed with pneumonia, they typically appear with a severe disease. A number of symptoms, such as fever, chills, tachypnea, cough, lower chest indrawing, and chest or even abdominal pain, are frequently observed in clinical settings. Fever was the most sensitive sign in a Canadian series of 570 pediatric patients with signs and symptoms that suggested pneumonia. Grunting and retractions were the most specific signals, and they were related with alveolar infiltrates on a chest x-ray (CXR). The series was conducted in Canada. During the course of a recent study, hospital-based studies of pneumonia in children aged 0 to 59 months were examined. The study found that more than 80 percent of the cases involved CXR screening. For the purpose of diagnosis, the presence of "dense opacity occupying a portion or the entire lobe of the lung or presence of a pleural effusion on CXR" was the primary reference. Age-based tachypnoea had a sensitivity of 0.92 and a specificity of 0.22 for the diagnosis of radiographic-confirmed pneumonia, while chest indrawing had a sensitivity of 0.74 and a specificity of 0.15 for the same purpose. Additionally, out of the ten studies that were included, 24.9% (n = 3743) had CXR "confirmed" pneumonia. When compared to the clinical manifestations of pneumonia in older children, the clinical manifestations of pneumonia in infants and young children are often distinct. There is a possibility that infants who are in their first three months of life will only appear with a cough and respiratory distress when they have a low-grade fever or none at all.[44] At first glance, the clinical manifestations of Hib-associated CAP are comparable to those of other common bacteria; however, the beginning of the condition is typically more subtle. Infection with atypical microorganisms, such as M. pneumoniae, is typically associated with a

more slow clinical start that is accompanied by a variety of symptoms. These symptoms include headache, malaise, nonproductive cough, and low-grade fever or no temperature at all. [45,46]

The "Revised WHO Classification and Treatment of Childhood Pneumonia at Health Facilities" (2014) has provided guidelines for the clinical diagnosis and management of childhood pneumonia. These guidelines were developed in response to the wide variety of approaches that are taken to diagnose pneumonia. The goal of these guidelines is to ensure prompt diagnosis even in settings with limited resources, and they are based solely on clinical characteristics. [47]

WHO MODIFIED ARI CRITERIA 2014 [47]



Physical Findings

Symptoms	Grading
Fast breathing , with or without chest indrawing, no danger sign, normal saturation RR > 60/min below 2 months RR > 50/min between 2-12 months >40/min in children aged 1-5 years	Pneumonia
Lower chest indrawing, danger signs present (unable to drink or breastfeed, convulsions, lethargy, unconsciousness, severe respiratory distress, central cyanosis)	Severe Pneumonia

According to the World Health Organisation (WHO), the most important clinical symptom that is utilised to diagnose pneumonia is the presence of age-specific tachypnea or lower chest indrawing. Pneumonia is diagnosed in children who exhibit tachypnea or lower chest indrawing, as stated by the most recent guidelines established by the World Health Organisation (WHO). Children who exhibit a danger indicator, such as an inability to drink, recurrent vomiting, convulsions, lethargy, impaired level of consciousness, stridor, or severe malnutrition, are considered to be suffering from severe pneumonia. The easiest technique to determine the child's respiration rate is to observe them for a period of sixty seconds when they are calm. While there are a number of other respiratory symptoms that could be indicative of pneumonia, no single symptom can be used to diagnose or rule out pneumonia on its own.[48]

The respiratory rate appears to produce higher agreement amongst observers than auscultation of the chest, particularly when it comes to newborns. There is a respiratory rate of sixty breaths per minute in newborns younger than two months of age, fifty breaths per minute for infants between two and twelve months of age, and forty breaths per minute for children between one and five years of age, according to the World Health Organization's classification of tachypnoea. Although tachypnea is not specific, it is typically more sensitive than crackles on auscultation. As a result, it is extensively used to identify chronic obstructive pulmonary disease (CAP) in low- and middle-income countries (LMICs), where pneumonia is extremely common and resources are limited. [49]

Radiological Findings

In general, chest x-rays are considered to be standard procedure when it comes to hospitalised children who are being evaluated for a possible diagnosis of pneumonia. It is not consistent among studies whether clinical signs and symptoms at the time of a chest x-ray may accurately predict a radiological diagnosis of pneumonia, particularly in instances that are not considered to be severe. An additional essential factor to take into consideration is the fact that the interpretation of CXR findings is contingent on the quality of the video as well as the level of experience possessed by the reader. [50,51]

In addition, most guidelines do not suggest the frequent use of CXRs for children older than two months of age who are being cared for in outpatient settings. This is due to the fact that CXRs do not alter the outcome of upper respiratory tract infections (LRTIs). Because studies have produced contradictory findings, the question of whether or not the addition of a lateral CXR improves accuracy is a contentious one. In the WHO criteria for the standardisation of CXR interpretation,

primary end-point pneumonia was defined as consolidation, which is defined as "a dense opacity that may be a fluffy consolidation of a portion or whole of a lobe or of the entire lung, often containing air bronchograms." Additionally, consolidation was defined as interstitial infiltrates with a CRP that was greater than 40 ng/mL or pleural effusion. It has been reported that such an approach improves agreement. [48,52]

The level of agreement was lower when comparing the presence of any infiltrate (as the end-point) to other infiltrates that did not satisfy this description. Sixty It is possible that radiographic abnormalities that are consistent with pneumonia are present in a sizeable number of children younger than five years old who are experiencing fever and leukocytosis and who do not have a clearly identified source of infection. Twenty-six percent of the patients younger than five years old who reported to the emergency room with fever, leukocytosis larger than 20,000 cells/mm3, and no clinical symptoms suggestive of pneumonia received a confirmed diagnosis of pneumonia on CXRs, according to a study that was conducted by Bachur and colleagues. The evaluation of nonspecific clinical indications of infection in this age group therefore includes a plain CXR as one of the aspects to be investigated. [53]

Computed tomography (CT) scans should not be utilised routinely unless another underlying illness is suspected (for example, a tumour or abscess). This is due to radiation concerns, as well as the fact that less invasive imaging modalities typically serve for diagnosis and management. There is the possibility of an exception being made in situations when a surgical intervention is being considered, such as in the case of complex empyema situations. [54,55]

Additionally, a comparison between conventional chest radiography and lung magnetic resonance imaging (MRI) has been attempted in a few publications. It has been stated that there is a high level of agreement pertaining to the increased

sensitivity of magnetic resonance imaging (MRI) for consequences such as lung abscess and necrosis, along with the benefit of being a radiation-free method. [56]

MANAGEMENT

Choice of Empirical Antibiotic Treatment for Community-Acquired Pneumonia According to Age and Clinical Picture:

Age/Clinical Picture	Inpatient	Outpatient	Guideline Recommendation
Newborn	I.V. Ampicillin + I.V. gentamicin. Consider adding Azithromycin[a]	—	1. AJTCCM 2020
1 month to 5 years	I.V. Penicillin or I.V. Ampicillin[a] (1, 5) I.V. Ampicillin (or penicillin) and Gentamicin (2) I.V. Amoxacillin-clavulanate[a] (3, 4) Children with life-threatening illness: Third-generation I.V. cephalosporin, consider coverage for MRSA (2, 5)	Oral Amoxicillin[a,b] (1, 2, 3, 4, 5)	1. IDSA 2011, BTS 2011, CIDS/CTS 2000 2. WHO 2014, 2018 3. AJTCCM 2020 4. NICE 2019 5. Canadian 2018
5 years and older: alveolar infiltrate, pleural effusion	I.V. Penicillin or I.V. Ampicillin; add macrolide if not responding (1) Children with life-threatening illness: Third-generation I.V. cephalosporin, consider coverage for MRSA (1, 3)	N/A	1. IDSA 2. WHO 2014, 2018 3. Canadian 2018
5 years and older: interstitial infiltrate,	I.V. Ampicillin (1, 2, 3); consider adding a β-lactam if not responding (1) Macrolides if findings of atypical community-acquired pneumonia.	Amoxicillin (1,[a] 2, 3)	1. IDSA 2. WHO 2014, 2018 3. Canadian 2018

Choice of Antibiotic Treatment for Community-Acquired Pneumonia When Typical Bacteria Are Identified:

Pathogen	First Choice	Other
S. pneumoniae, penicillin susceptible or intermediate	Penicillin, ampicillin, amoxicillin	Cefuroxime, ceftriaxone
S. pneumoniae—penicillin resistant (MIC ≥4 μg/mL)	Second- or third-generation cephalosporins for sensitive strains; vancomycin	Levofloxacin, linezolid
S. aureus	Methicillin/oxacillin (for MSSA)	Vancomycin or teicoplanin (for MRSA), linezolid
H. influenzae	Amoxicillin/clavulanate	Cefuroxime, ceftriaxone, other second- and third-generation cephalosporins
M. catarrhalis	Amoxicillin/clavulanate or cefuroxime	Azithromycin, cefprozil

ROLE OF MICRONUTRINETS IN PNEUMONIA

Zinc and Vitamin A

Zinc and vitamin A are two micronutrients that are crucial to the body's defence mechanisms. If one of these micronutrients is lacking, it may be more challenging for

the immune system to successfully fight off infections[57,58]. Despite the fact that both of these nutrients have been shown to have favourable effects on immune function[59-61], there have been relatively few studies that have directly investigated the therapeutic benefits of these micronutrients specifically in the context of paediatric CAP.

Vitamin D Supplementation

Both phagocytosis-dependent and antibody-dependent macrophages are stimulated by vitamin D, which results in the development of antimicrobial capabilities. T and B cells are also affected by 1,25-dihydroxy vitamin D3, which has the ability to modify the actions of lymphocytes that are responsible for the production of cytokines and antibodies. A severe lack of vitamin D can cause deformities and hypotonia in the chest wall, which in turn can result in decreased lung volume, poor compliance of the chest wall, atelectasis, and fibrosis. A study conducted in Ethiopia discovered that children who were diagnosed with pneumonia had a thirteenfold greater frequency of severe vitamin D insufficiency. This was demonstrated clinically by the presence of rickets.[62]

VITAMIN A

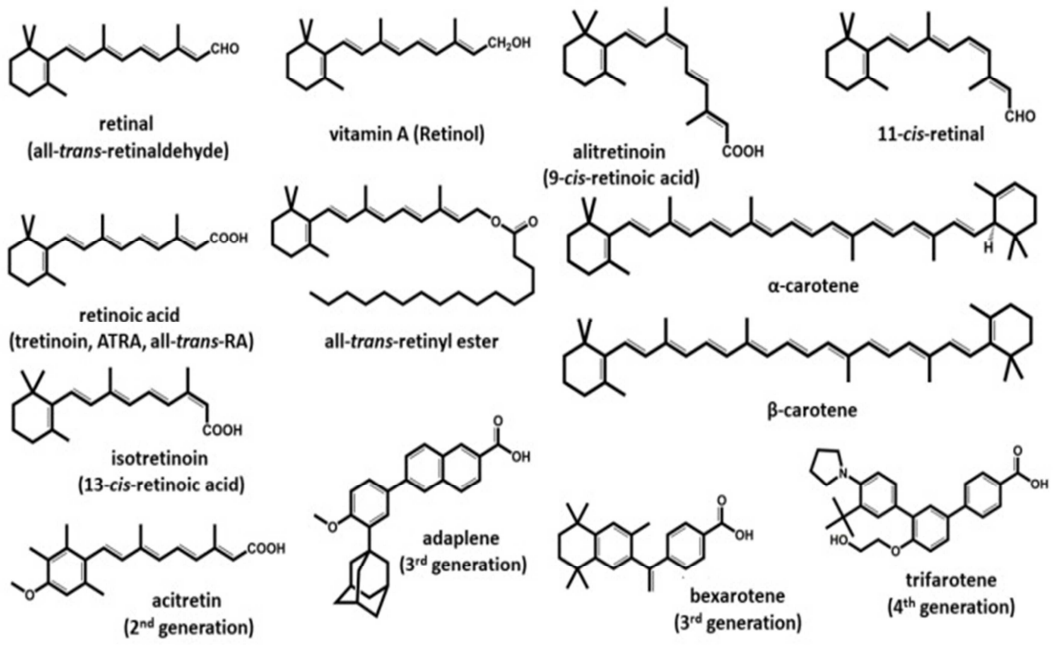
Vitamin A Deficiency (VAD) is the major cause of preventable childhood blindness among children living in countries with lower and intermediate incomes. It also has a significant role in the morbidity and death rates of children that are caused by infectious infections. There is a correlation between VAD and an increased risk of contracting a variety of ailments, such as diarrhoea, measles, and respiratory infections. There are around one third of preschool-aged children around the world who are deficient in vitamin A. The majority of these children reside in South East

Asia, and India is home to the highest number of children who suffer from vitamin A deficiency. Similarly, India is home to the biggest number of youngsters who are below the clinical threshold for vitamin A deficiency.[63]

Vitamin A deficiency (VAD) has been linked to histological alterations in the pulmonary epithelial lining. These alterations impair the normal physiology of the lungs, making the patient more susceptible to severe tissue dysfunction and respiratory illnesses.[64] Consequently, a subclinical form of VAD might result in serious complications such as pneumonia.[65] Vitamin A supplementation resulted in a considerable reduction in the amount of time required for the symptoms to disappear in children whose serum retinol levels were normal. These adverse changes were almost reversed once the levels of vitamin A were restored to the desired level, which confirmed the efficacy of vitamin A in the preservation of organ architecture and cell differentiation during the growing period. This finding also provided a justification for the therapeutic vitamin A supplementation programs that are currently being implemented.[64]

There is a family of lipid-soluble chemicals known as retinoic acids, and vitamin A is a subclass which belongs to that family. These are made up of four isoprenoid units that are connected together in a pattern that is head-to-tail. Preformed vitamin A and provitamin A carotenoids, which include beta-carotene and other forms, are the two primary sources of vitamin A. Carotenoids, which resemble provitamin A, can be found in plants. While beta-carotene, alpha-carotene, and beta-cryptoxanthin are the most common forms of provitamin A, there are numerous more types as well. Mammals are capable of converting them into vitamin A, but with varied degrees of metabolic efficiency. The most active form of vitamin A is called preformed vitamin A, which includes retinol, retinal, retinoic acid, and retinyl esters.

This form of vitamin A is mostly found in dietary sources that come from animals, and it is also the type that is provided by the majority of supplements. The combination of provitamin A (also known as beta-carotene) and preformed vitamin A is that which is provided by certain supplements. [66]



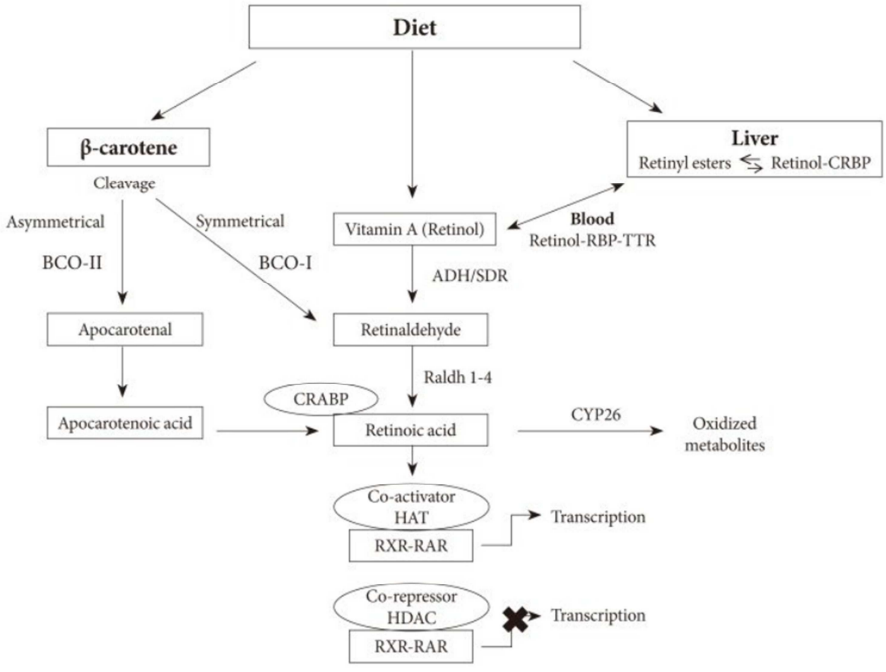
SOURCES: Stem cells, liver, kidneys, egg yolks, and butter are the most prevalent foods that contain preformed vitamin A, also known as retinols. Generally speaking, green leafy vegetables, sweet potatoes, and carrots are the best sources of provitamin A, also known as beta-carotene. Vitamin A that comes from animal sources or supplements is more likely to induce toxicity than provitamin A that comes from plant sources. This is due to the fact that vitamin A is preformed.[66]

In order to tackle this major health problem, the government of India through its national immunization schedule is supplementing Vitamin A to all children during the course of routine immunization .

Vitamin A Schedule according to National Immunization Schedule (NIS) [67]

Vaccine	When to give	Dose	Route	Site
Vitamin A (1st dose)	At 9 completed months with measles-Rubella	1 ml (1 lakh IU)	Oral	Oral
Vitamin A (2nd to 9th dose)	16-18 months. Then one dose every 6 months up to the age of 5 years.	2 ml (2 lakh IU)	Oral	Oral

Metabolism and various metabolic forms of Vitamin A [68]



CAUSES AND RISK FACTORS[69]

Causes

- Dietary lack of vitamin A due to any of the following:
    - In low- and middle-income countries, poor availability of vitamin A–rich foods such as:
        - Brightly colored fruits and vegetables such as melons, carrots, sweet potatoes, and tomatoes
        - Liver, milk, and eggs
    - Vegan diet
    - Eating disorder causing deficiencies of multiple micronutrients including vitamin A
- Increased demand for vitamin A occurring particularly during pregnancy [3]
- Vitamin A malabsorption and reduced/absent ability to convert carotene to active vitamin A, which commonly occurs in fat-malabsorptive conditions such as:
    - Inflammatory bowel disease (eg, Crohn disease)
    - Pancreatic insufficiency
    - Short bowel syndrome
    - Cystic fibrosis
    - Celiac disease
    - History of certain types of bariatric surgery, such as Roux-en-Y gastrointestinal bypass surgery

Risk factors and/or associations

Age

- Children are at higher risk than adults for dietary vitamin A deficiency

Sex

- Females are at elevated risk for dietary vitamin A deficiency

Genetics

- Single nucleotide variants in 12 genes are associated with retinol and beta carotene levels and with beta carotene bioavailability
- Populations with different allele frequencies for these single nucleotide variants may differ in ability to absorb dietary beta carotene and thus in risk for vitamin A deficiency

Other risk factors/associations

- Most cases of vitamin A deficiency due to lack of vitamin A in diet occur in the following geographic areas:
    - Southeast Asia
    - Sub-Saharan Africa
- Incidence of vitamin A deficiency due to lack of vitamin A is high among refugees and populations under economic stress
- End-stage liver disease due to conditions such as alcoholic cirrhosis and hepatitis C

DEFICIENCY

It is estimated that roughly thirty percent of children under the age of five around the world are deficient in vitamin A, and that almost fifty percent of young children in South Asia and sub-Saharan Africa are similarly affected[70]. Malnourished children and adults are more likely to experience night blindness, full blindness, and

later stages of xerophthalmia in many countries with insufficient resources[71,72]. Some of these conditions include complete blindness. Each year, around 500,000 preschool-aged children are diagnosed with blindness, and a significant number of them pass away[73]. This is a significant public health issue. The prevention of these ocular problems can be achieved through the routine delivery of vitamin A to children living in endemic areas[74,75]. Furthermore, according to the findings of a study[76], administered vitamin A supplements to children who are part of undernourished populations have the potential to lessen the long-term risk of hearing loss among those who have ear discharge, which is a sign of otitis. When vitamin A is routinely administered to children in locations where the disease is endemic, there is a slight reduction in the mortality rate of children, which ranges from around 5 to 15 percent[70]. This was demonstrated in a cluster-randomized experiment that was carried out in a competent manner and was called the DEVTA trial. The trial involved more than two million children in North India who were between the ages of six months and five years old. This population has a significant prevalence of vitamin A deficiency[77]. Although supplementation with high-dose vitamin A (200,000 international units every six months, with or without a deworming protocol) for a period of five years resulted in a reduction in the prevalence of severe vitamin A deficiency and ophthalmopathy, it did not result in a significant reduction in mortality (mortality ratio 0.96, 95% confidence interval 0.89-1.03). In contrast to the findings of earlier, more limited research, which revealed that taking vitamin A supplements on a regular basis led to a decrease of 12 percent in mortality from all causes and a reduction of a similar magnitude in mortality connected with diarrhoea[78], our discovering contradicts those findings. The DEVTA and eight other studies were included in a meta-analysis that resulted in a weighted average mortality decrease of 11 percent (95% confidence interval: 5-16) [77].

Patients with disorders associated with fat malabsorption, such as cystic fibrosis and other causes of pancreatic insufficiency, coeliac disease, cholestatic liver disease such as primary biliary cholangitis, small bowel Crohn disease, short bowel syndrome, and patients who have undergone certain types of bariatric surgery, can also be affected by vitamin A deficiency, with or without xerophthalmia. This condition can also be observed in resource-rich countries in patients who have undergone certain types of bariatric surgery. [79]

VITAMIN A DEFICIENCY AND THE LUNG

The development of a human lung begins during the fifth week of foetal development and continues during the first several years of growth. The embryonic phase, the pseudoglandular phase, the canalicular phase, the saccular phase, and the alveolar phase are all included in it. After birth, the foetal lung develops into one of the most complex organs, with roughly forty distinct cell types. This organ is characterised by its complexity. The fundamental purpose of the lungs is to fulfil the requirements of the organism for the elimination of carbon dioxide and oxygen. This takes place in the alveoli of the lungs, which are created in part by the subdivision (septation) of the gas-exchange saccules of the embryonic lung. There are significant differences between species in terms of the developmental stage at which septation takes place. The timely process of lung differentiation, which results in the formation of alveolar structures, mostly takes place during the third trimester of pregnancy in human beings and continues in postpartum years, as well as throughout the first years of development. The process of lung maturation occurs during the perinatal period in a manner that is highly controlled, and interactions between the alveolar epithelial and mesenchymal layers make a significant contribution. The hepatic stellate cells of the liver, are also present in other organs, are sites where vitamin A is stored. Despite the

fact that the quantity of retinol in them is significantly lower than that of the liver, these cells, which have also been found in the lungs, including the foetal lung, have the ability to take up retinol from chylomicron. This suggests that this cell gets it by a mechanism that is comparable to that of the liver. When there is an elevated need for retinol by this tissue, particularly in the growing lung, when the morphology of the lungs is still immature, the primary function of RE reserves in the lung is to guarantee that direct retinol administration is carried out. In light of this, the supply of vitamin A from the mother is of critical significance for ensuring appropriate foetal nourishment, growth, and development. During the maturation of the lungs and the postnatal period, these reserves serve as the foundation for the production of RA. Furthermore, the synthesis of RBP that occurs during foetal and neonatal development is not sufficient to provide a continual supply from the liver's storage. Deficiencies in the dietary requirements of the growing lung while the foetus is still in the womb provide a threat to the integrity of the respiratory system. In the process of controlling early lung growth and alveolar formation, vitamin A plays a significant role through the creation of RA, which is associated to this process. Therefore, it is important to ensure that enough intake is maintained during the final month of pregnancy. This will ensure that retinyl reserves are present in the developing lung, which is necessary for the production of retinol during lung maturation and postnatal life. RA is responsible for regulating cell proliferation and differentiation, as well as proper organogenesis, during the embryonic development process. The low vitamin A status of the newborn appears to be a contributing factor in the risk of bronchopulmonary dysplasia (BPD), which is a chronic lung disease that is characterised by focal loss of ciliated cells, keratinising metaplasia, and necrosis of the bronchial mucosa, in addition to an increase in mucous-secreting cells. This becomes even more significant in premature infants, as their serum retinol and RBP levels are significantly lower than those of

full-term neonates. In point of fact, abnormal lung function, decreased alveolar number, decreased protection against infections, and a higher probability of developing acute illnesses in childhood and chronic illnesses in adulthood, including the risk of lung cancer, have been linked to low liver stores in neonates and a low supply during lactation and have been linked to abnormal lung function. [80]

In addition, during the postnatal period, RA is necessary for the growth of the lungs, the alveolarization of the lungs, and the expression of the primary components of the extracellular matrix (ECM), which plays a significant role in the resistance and elasticity of the lungs with regard to repair and remodelling. As a consequence of this, VAD will cause significant alterations in the structure and function around the lungs. In human beings, endemic VAD is linked to a poor forced vital capacity (FVC), which is a measure of airway blockage and a powerful predictor of mortality in individuals who are asymptomatic and do not have chronic respiratory diseases. When McCollum first described "fat-soluble A," which was subsequently determined to be retinol, he made the observation that animals that were artificially deficient in this component "have frequently suffered from prevalent bronchitis." This observation was made in 1913. It would appear that keratinisation of the tracheae and bronchi takes place quite quickly in patients with VAD, in fact, appearing before changes in the eye.[80]

Respiratory Infections[80]

Every year, acute respiratory infections, most commonly pneumonia and influenza, are responsible for the deaths of more than four million people over the world. Furthermore, when viewed in a global context, the death rate that is solely attributed to these diseases is ten times greater than the worldwide median death rate that is attributed to all causes. The anti-infective properties of vitamin A were

established by Green and Mellanby in the year 1928. Vitamin A has been shown to be an immune-modulating agent and to play a significant part in the immune system's response to infections, according to a number of studies. According to these findings, VAD makes a significant contribution to the morbidity and mortality rates of children, with the epithelia of the trachea and respiratory tree being among the first tissues to exhibit histological alterations. Previous research conducted by Sommer and colleagues demonstrated that "the risk of respiratory disease and diarrhoea were more closely associated with vitamin A status than with general nutritional status". When it comes to protecting animals against diseases, the epithelial tissues serve as the initial line of defence. Several of the effects of undernutrition are mediated by the immune system. These effects include changes in the host's defences, which can reduce the host's ability to recover from diseases or increase their resistance to infections. Additionally, VAD is responsible for squamous metaplasia of the respiratory epithelium, which is characterised by the replacement of ciliated epithelial cells with squamous epithelium. This condition also leads to a reduction in the production of mucus. All of these are elements that have the potential to raise the probability of invading infections. The function of residents macrophages, neutrophils and natural killer cells and the development of T-cells mediated antibody responses are also impaired in VAD, leading to a decreased protective mechanism at mucosal surfaces. During embryogenesis, maternal RA is essential for the development of secondary lymphoid organs, whereas throughout the adult life RA controls the differentiation of immune cells necessary for immune tolerance via Treg induction in in vitro and in vivo animal models.[80]

In people who have lower amounts of vitamin A in their plasma, they are more likely to experience recurrent respiratory infections. This is one of the most significant health issues that are prevalent in underdeveloped nations. In addition, when viral disorders, particularly those that affect the respiratory system, are present, plasma retinol levels decrease. This, in turn, leads to a higher vulnerability to infection, which creates a "vicious circle." One possible explanation for this phenomenon is that acute infections are accompanied by an increase in the metabolic requirement and/or an increase in the renal clearance of retinol and RBP. Infants in developing nations are given vitamin A supplements, and it is generally acknowledged that these supplements are healthy and contribute to a reduction in respiratory infections. However, this is not always the case, and supplementing is still a contentious topic. [80]

Some other supplementing trials did not find any evidence that the supplement had a favourable effect in regions where the prevalence of VAD was high. The distinct response of supplementation against certain pathogens, or its differential effects depending on the nutritional status, or the most recent vaccines given, the sex and age of the child, the season, or the differential effects of RA on target cells, may be able to explain, at least in part, the clinical results that are contradictory against one another[80].

LITERATURE FROM PREVIOUS STUDIES:

In the comprehensive research that Mendes et al. (2022) conducted, they looked on the prevalence of vitamin A deficiency (VAD) in children who were hospitalised with pneumonia and were between the ages of 6 months and 5 years old. According to the findings of the study, which analysed ten publications that were eligible for inclusion from a variety of databases, all of the studies indicated subclinical VAD levels that were greater than twenty percent, with the highest level

reaching ninety-two percent. It was determined that this impairment has a significant role in immunological function and epithelial integrity, which makes it a key contributor to an increased risk of the respiratory illness pneumonia. A low socioeconomic status, inadequate sanitation, environmental pollution, a lack of breastfeeding, and incomplete vaccination are some of the risk factors that contribute to the severity of pneumonia. Both the maintenance of epithelial barriers and the modulation of immunological responses require vitamin A as an essential component. Both the activity of phagocytic cells and the markers of "Natural Killer" (NK) cells, which are very important for immunological defence, are supported by this substance. As a result of VAD's ability to impair these defences, the risk of respiratory infections such as pneumonia is increased. The findings of this study highlight the high frequency of VAD in children who are hospitalised with pneumonia, hence highlighting the necessity of implementation of targeted therapies. Supplementation with vitamin A is an efficient and cost-effective method for lowering the morbidity and death rates among children. This is accomplished by enhancing immunity and lessening the severity of infections. The findings underscore the significance of incorporating dietary programs into public health strategies, particularly in locations with low resources, in order to enhance the health outcomes of children and to combat mortality that are caused by pneumonia.[81]

In the research paper titled "Association Between Serum Vitamin A Levels and Recurrent Respiratory Tract Infections (RRTIs) in Children," Wang et al. (2022) carried out a cross-sectional study in Beijing, encompassing a total of 8,034 children and adolescents ranging in age from six months to seventeen years. A link between serum vitamin A levels and the incidence of recurrent respiratory tract infections (RRTIs) was the focus of this investigation. Based on the data, it was determined that

721 children (8.97%) were diagnosed with vitamin A deficiency, while 3,073 children (38.25%) were found to have subclinical vitamin A deficiency. Particularly noteworthy is the fact that only 28.8% of children with vitamin A insufficiency and 53.1% of children with subclinical deficiency had no RRTI or RTI symptoms. This suggests that a sizeable section of the population may be suffering from deficiencies that have not been recognised due to the absence of excessive symptoms. In addition, the research showed that children who lacked vitamin A had a chance of acquiring recurrent respiratory tract infections (RRTIs) that was 6.924 times higher than the risk of children who had normal levels of vitamin A. The incidence of recurrent respiratory tract infections (RRTIs) was 2.140 times higher in children who had a subclinical vitamin A deficit. It is important to note that these correlations remained even after taking into account any confounding factors, which highlights the significant role that adequate amounts of vitamin A play in maintaining respiratory health. Both the maintenance of epithelial integrity and the modulation of immunological responses require vitamin A as an essential component. This study underlines the need for public health initiatives that aim to improve vitamin A status among children and adolescents. The high incidence of both clinical and subclinical vitamin A deficiencies that was discovered in this study highlights the need for improved vitamin A status. The findings of the research conducted by Wang et al. indicate that there is a strong correlation between low serum vitamin A levels and the occurrence of recurrent respiratory tract infections (RRTIs) in children. It is possible that addressing vitamin A insufficiency in this population by dietary adjustments or supplementation could be a promising method for reducing the prevalence of recurrent respiratory infections. [82]

Among Indian children aged 12–59 months, the research conducted by Kundu et al. (2021) makes use of information obtained from the Comprehensive National Nutrition Survey (CNNS) to investigate the prevalence of Vitamin A Deficiency (VAD) and the factors that contribute to its development. According to the data, the prevalence of VAD is 17.54% across the board, with statistically significant differences being linked to socio-economic and dietary determinants. According to the findings, the prevalence of VAD was higher in children who came from economically poor households. In particular, those who were in economically disadvantaged portions had a higher prevalence of VAD in comparison to their peers who were in affluent sectors. This highlights the impact that economic discrepancies have on the nutritional condition of individuals. Diverse dietary intake was found to be a key factor in the development of VAD. The fact that children with a limited variety of foods consumed were more likely to be affected by VAD highlights the significance of consuming a wide range of foods in order to guarantee an appropriate intake of vitamin A. A higher prevalence of vitamin A deficiency was found in children who were stunted, which is an indication of chronic undernutrition. This finding suggests that there is a connection between overall nutritional status and vitamin A levels. Education of mothers was a significant factor in the occurrence of vitamin A deficiency (VAD) among offspring. There was a correlation between higher levels of maternal education and a lower prevalence of VAD, which highlights the impact that mother knowledge and behaviours have on the nutrition of children to a greater extent. The study also discovered that children who were breastfed for longer periods of time had a reduced prevalence of VAD. This finding suggests that prolonged nursing may play a preventive effect against vitamin A deficiency. The results of this study underscore the necessity of focused nutrition programs that address socio-economic gaps and encourage diversified dietary choices in order to

minimise the prevalence of dietary-related diseases among children in India. increasing maternal education, increasing economic conditions, and supporting prolonged breastfeeding are all strategies that should be implemented in order to effectively treat VAD and improve the health outcomes for children. [83]

Gultom et al. (2020) carried out a study that was cross-sectional in nature in order to evaluate the connection between a lack of vitamin A and the occurrence of pneumonia in children under the age of five in the province of West Java in Indonesia. According to the findings of the study, which analysed responses from 594 youngsters, secondary data from the 2017 Indonesian Demographic and Health Survey was utilised. The occurrence of pneumonia, which was the dependent variable, and the status of getting vitamin A supplements, which was the independent variable, were the key variables that were investigated. Structured questionnaires were used to gather data, and chi-square tests were used to analyse the data in order to determine the extent of the relationships between the variables. Based on the findings, it was determined that 38.6% of the children had not been given vitamin A supplements, and 26.8% of them had reported having experienced pneumonia. Children who did not receive vitamin A supplementation had a slightly greater incidence of pneumonia (27.0%) compared to children who did receive supplementation (26.7%). The investigation, on the other hand, established that an insufficient intake of vitamin A was associated with an increased risk of pneumonia; however, this link did not meet the criteria for statistical significance (Odds Ratio [OR] = 1.011; 95% Confidence Interval [CI]: 0.690 to 1.481; p = 1.000). The findings of the study indicate that although there is a slight increase in the risk of pneumonia among children who do not receive vitamin A supplementation, the lack of statistical significance suggests that other factors may play more significant roles in the occurrence of pneumonia.

The socioeconomic situation, environmental conditions, access to healthcare, and overall nutritional status are all examples of several aspects that could be considered. The authors agree that the significance of vitamin A in preventing respiratory infections is still up for debate. This is consistent with the findings of prior research that have yielded contradictory findings about the effectiveness of vitamin A supplementation in lowering the morbidity and death rates associated with respiratory infections. In conclusion, although this study did not discover a statistically significant link between vitamin A supplementation and a reduction in the incidence of pneumonia, it does highlight the significance of continuing research in order to uncover interventions that are successful. It is vital to implement comprehensive strategies that address a variety of factors that influence the health of children in order to reduce the risk of pneumonia in this vulnerable population. [84]

An investigation was carried out by Yi-Ling J et al. (2016) with the purpose of determining whether or not there is a correlation between the serum level of vitamin A (VA) and the severity of pneumonia and recurrent respiratory infection (RRI) within one year of treatment in children who were diagnosed with pneumonia. Additionally, the researchers wanted to establish a foundation for the utilisation of serum VA level as an index for evaluating the condition of pneumonia and predicting the likelihood of recurrent respiratory infection. A total of 88 infants under the age of three who were diagnosed with pneumonia were recruited to participate in the research study. At the time of hospitalisation, the serum VA level was determined, and the development of RRI was monitored over the phone within a given year after the patient was discharged. The children who were diagnosed with pneumonia had a decrease in the serum level of VA, which registered at 0.8±0.3 μmol/L. A significant difference was seen between the severe pneumonia group and the mild pneumonia

group in terms of serum levels of vitamin A deficiency (VAD) (0.7±0.3 μmol/L versus 0.9±0.3 μmol/L; P<0.05). Furthermore, the severe pneumonia group exhibited a substantially greater detection rate of vitamin A deficiency (VAD) compared to the mild pneumonia group (63% versus 28%; P<0.05). Over the course of one year, the youngsters were monitored. There were no significant differences in the incidence of RRI between the group with suspected subclinical vitamin A deficiency (SSVAD)-pneumonia and the group with normal VA-pneumonia, nor between the group with VAD pneumonia and the group with SSVAD-pneumonia (P>0.05). However, the group with VAD pneumonia showed a significantly higher incidence of RRI than the group with normal VA-pneumonia, with 49% for the former and 18% for the latter. [85]

An investigation of the connection between deficiencies in vitamins A and E and the prevalence of viral disorders in children was carried out by Y.-J. Qi and colleagues in the year 2016. There were 684 healthy children who participated in the study. Their ages ranged from 5 months to 12 years. It was determined through the use of high-performance liquid chromatography that serum levels of vitamins A and E were measured by collecting blood samples under settings that protected them from light. Instances of acute respiratory tract infections (ARI) and diarrhoea were documented among the subjects two weeks after the sample was completed. According to the findings, children's susceptibility to developing acute respiratory infections (ARI) and diarrhoea was increased when they lacked vitamin A. To be more specific, the proportion of children who had diarrhoea and also had lowered blood vitamin A levels was higher than the proportion of children who did not have diarrhoea. This finding suggests that a vitamin A level that is lower than 0.2 mg/L is more likely to be related with acute respiratory illness (ARI). Additionally, a lack of vitamin E was associated with a higher prevalence of acute respiratory infections

(ARI) in the youngsters. Following the findings of the study, it was determined that deficits in vitamins A and E are significant factors that are associated with the occurrence of acute infectious illnesses in children. The findings of this study highlight the significance of ensuring that adequate levels of these vitamins are maintained in order to improve immune function and lower the risk of illnesses such as acute respiratory infections (ARI) and diarrhoea. [7]

In order to evaluate Vitamin A Deficiency Disorders (VADD) among rural pre-school children in South India, Nimmathota Arlappa, N Balakrishna, and their colleagues carried out a comprehensive study. The study involved the examination of 35,480 children between the ages of one and five years old. A cross-sectional design with multi-stage random sampling was utilised in the research project that was carried out by the National Institute of Nutrition. This was done to ensure that the study was representative of the population. 59.3 percent of the children were found to have sub-clinical VAD, which is a prevalence level that is regarded to be a significant public health hazard according to the guidelines of the World Health Organisation. In addition, 0.6% of children demonstrated clinical forms of VAD, which further emphasises the pervasive extent of the deficit. There were considerable socio-demographic differences that were brought to light by the study. The study found that children from marginalised communities, households with lower incomes, and children whose mothers were illiterate had a greater prevalence of VAD. A lack of food diversity and limited access to medical care were both factors that contributed to the problem. The findings bring to light the critical requirement for focused interventions, such as increasing the consumption of foods that are rich in vitamin A and continuing to implement high-dose vitamin A supplementation programs on a consistent basis. In order to address the underlying causes of VAD, public health

programs should place an emphasis on improving sanitation, educating mothers about nutrition, and raising awareness about maternal health. The findings of this study highlight the significance of maintaining surveillance and intervention efforts in order to lower the illness and death rates among children in India's rural population that are connected with vitamin A deficiency.[63]

88 children under the age of three who had been diagnosed with pneumonia were included in the study that was conducted in 2016 by Jiang Yi-Ling and Peng Dong-Hong. The serum vitamin A levels of these children were analysed in order to investigate the association between vitamin A insufficiency and the severity of pneumonia. A study was conducted to determine that children who were diagnosed with pneumonia had a blood vitamin A level that averaged 0.8 ± 0.3 μmol/L. Particularly noteworthy is the fact that children with severe pneumonia display significantly lower levels in comparison to children with moderate pneumonia. According to the findings, vitamin A deficiency (VAD) was found in 63% of children who had severe pneumonia, but only 28% of children who had moderate pneumonia had VAD. Based on these findings, it appears that there is a significant correlation between decreased levels of serum vitamin A and an increased severity of pneumonia in young children. According to the findings of the study, adequate levels of vitamin A have the ability to reduce the severity of respiratory infections in individuals of this age group, which is particularly susceptible to them. [86]

According to the findings of the research conducted by Kathryn A. Thornton and colleagues, who investigated the correlation between Vitamin A Deficiency and Gastrointestinal and Respiratory Morbidity in School-Age, it was observed that children who had a Vitamin A deficiency of less than 10.0 μg/dL were at a higher risk of experiencing diarrhoea, along with vomiting, coughing, and fever. According to the

findings of this study, the concentrations of haemoglobin and vitamin A were found to have an inverse relationship with the rates of morbidity in children of school age. [87]

The authors of their research investigated "Low intake of vitamin A-rich foods among children, aged 12-35 months, in India: association with malnutrition, anaemia, and missed child survival interventions," Over the course of the 2005-2006 India National Family Health Survey, Richard D. Semba, Saskia de Pee, and their colleagues conducted an analysis of the data in order to determine the dietary intake of vitamin A-rich foods among young children and the association between that intake and health outcomes. Using a 24-hour dietary recall, the researchers were able to quantify the amount of vitamin A that was consumed by 17,847 children in India who were between the ages of 12 and 35 months. According to the data, 41.9% of these children did not consume foods that were rich in vitamin A throughout the period over which the recall was in effect. Particularly noteworthy is the fact that children who did not consume foods that were rich in vitamin A had higher rates of malnutrition indicators: 59.0% of them were stunted, whereas 52.5% of them consumed such foods; 32.9% of them faced severe stunting, whereas 26.7% of them did; 48.5% of them were underweight, whereas 43.8% of them were; and 21.6% of them experienced severe underweight, whereas 17.9% not did. There was a statistically significant difference between all of these differences ($P < 0.0001$). The level of education attained by the mother was found to be a significant factor: children whose moms had completed ten years of schooling or more were more likely to consume foods that were rich in vitamin A. A significant number of young children in India are not obtaining sufficient amounts of vitamin A-rich foods, which puts them at risk for malnutrition, anaemia, and the failure to receive necessary medical interventions. This is a significant public health concern that is brought to light by the study mentioned

above. Because of the substantial correlation between maternal education and the amount of vitamin A that children consume through their food, it is possible that increasing the education of women could have a significant role in strengthening the nutritional status and overall health of children. [88]

It is hence imperative that all children receive Vitamin A prophylaxis as per the national immunization schedule and be protected from fatal infections. There are no such similar studies in north Karnataka population addressing the issue. So this study is undertaken as it will estimate not only the prevalence of vitamin A deficiency in this population but also correlate its levels with the severity of pneumonia.

MATERIALS AND METHODS

Source of data: Children aged between 2 months and 60 months who are diagnosed with Pneumonia as per WHO criteria, and admitted in the Pediatrics Ward of KLES DR Prabhakar Kore Charitable Hospital, Belagavi

Methods of Collection of Data:

A. **Study Design:** Cross Sectional Observational Study
B. **Place of Study:** Paediatric Department, KLEH Dr. Prabhakar Kore Hospital, Belgaum, Karnataka
C. **Study Period:** 1 year (September 2023- August 2024)
D. **Sample Size:** The sample size for the current study was calculated using following equation [136]

Single Proportion – Absolute Precision

Expected Proportion of VA Deficiency = .51 (51%)[8]

Precision (%) = 10

Desired Confidence Level (%) = 95

Formula

$$N = \frac{Z^2 pq}{d^2}$$

Where n = Sample size = **96** Pneumonia cases

Z= Standard Normal Variable Value (Z=1.96 at 5% alpha error)

d = Margin of Error = 10%

p = 51%, q = 100 – 51 = 49%

Sampling technique: all children that meet inclusion criteria

INCLUSION CRITERIA:

All children between ages 2 months to 5years diagnosed with pneumonia according to revised WHO ARI criteria 2014 [47] -

Symptoms	Grading
Fast breathing , without chest indrawing or general danger sign, normal saturation RR > 60/min below 2 months RR > 50/min between 2-12 months >40/min in children aged 1-5 years	Pneumonia
Lower chest indrawing, danger signs + (unable to drink or breastfeed, convulsions, lethargy, unconsciousness, severe respiratory distress, central cyanosis)	Severe Pneumonia

EXCLUSION CRITERIA

Children with-

- Congenital Heart Diseases, congenital skeletal defects, congenital lung disease
- Immunodeficiency,
- Children with Allergic Airway Disease
- Patients using steroids or immune-suppressive drugs
- Children with Severe Acute Malnutrition
- Children whose parents/guardians did not provide consent for participation in the study

Institutional ethical Clearance:

The ethical clearance was obtained prior to the conduct of the study, from Institutional Ethics and Research Committee, KAHER's Jawaharlal Nehru Medical College, Belagavi.

C.T.R.I. registration: The study was registered with Clinical Trial Registry of India prior to sample collection and clearance was granted with registration number

CTRI/2023/05/067734.

METHODOLOGY

The current observational study was carried out in KLEH Dr. Prabhakar Kore Charitable Hospital, Belgaum, Karnataka from September 2023 to August 2024. After obtaining approval and clearance from the institutional ethics committee (Annexure - I), the patients fulfilling the inclusion criteria were enrolled for the study. Informed consent (Annexure - II) w ere obtained from parents of the infants after explaining to them the plan and intention of the study in the language they understood.

Total 96 patients meeting the inclusion criteria were included in the current study. Demographic Information regarding patients like age, sex, weight, area of residence and other clinical parameters were recorded.

Samples in 2ml plain vacutainer were collected and levels of serum Vitamin A were estimated by using ELISA.

Every piece of information was meticulously entered into an excel spread sheet and used for statistical analysis.

Specimen analysis - Samples were processed using BioAssay Technology Laboratory Vitamin A ELISA kit.

Reagents stored at 2-8°C.

- Standard Curve Range: 2ng/ml - 800ng/ml
- Sensitivity: 1.21ng/ml
- Size: 96 wells

Application and Principle of the Assay –

- The kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate is pre-coated with human VA antibody. VA present in the sample is added and binds to antibodies coated on the wells and then biotinylated human VA Antibody is added and binds to VA in the sample.
- Then Streptavidin-HRP is added and binds to the Biotinylated VA antibody. After incubation unbound Streptavidin-HRP is washed away during a washing step. Substrate solution is then added and color develops in proportion to the amount of human VA.
- The reaction is terminated by addition of acidic stop solution and absorbance is measured at 450 nm.
- Serum retinol measured in ng/ml, conversion factor applied and converted to micromole/litre

Specimen collection and processing -

Serum- allowed to clot for 10-20 minutes. Centrifuge at 2000-3000 RPM for 20 minutes.

All reagents, samples and standard are prepared as per specified instructions and assay is performed at room temperature.

Sample, reagent added to each well and incubated for 1 hour. Plate is washed 5 times. Substrate solutions A & B added, incubated for 10 minutes at 37 C.

Stop solution added, color develops, and result is read in 10 minutes.

Based on the serum Vitamin A level, participants were categorized into having normal level, subclinical deficiency and severe deficiency according to WHO guidelines.

>0.70 micromol/L	NORMAL
<0.70 micromol/L	SUBCLINICAL DEFICIENCY
<0.35 micromole/ L	SEVERE DEFICIENCY

STATISTICAL ANALYSIS:

The continuous variables were expressed as Mean ± SD or Median [IQ] and categorical variables were expressed as N (%).

To determine the normality of the clinical parameters on enrolment and follow-up, Shapiro-Wilk's test was performed (ANNEXURE III).

Statistical significance was estimated using chi-square test or test for proportions as appropriate.

For intra-group comparisons, independent t-test was performed when the data was normally distributed, and Mann-Whitney U test was performed when the data distribution was skewed.

For intergroup comparison, dependent t-test was performed when the data was normally distributed, and Wilcoxon matched pairs test was performed when the data distribution was skewed.

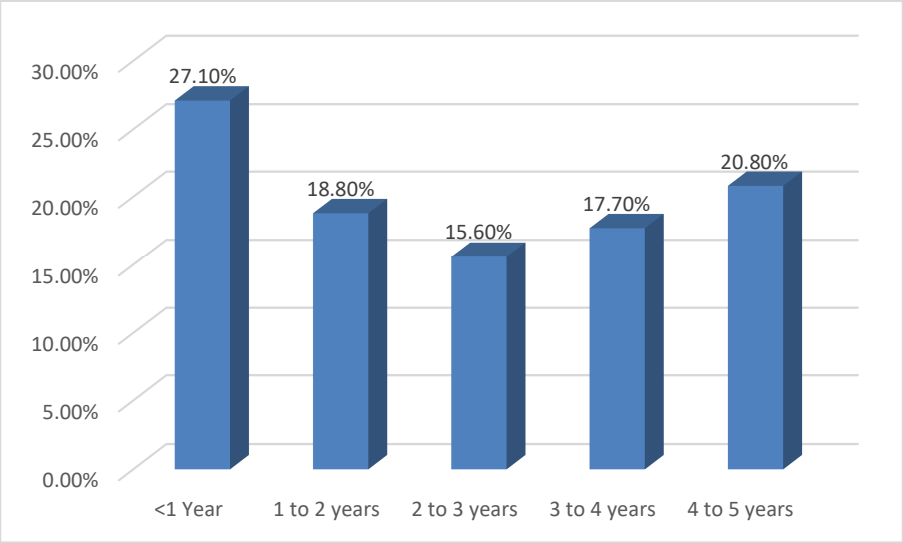
Correlation analysis was performed to understand the association between Vitamin A level and severity of Pneumonia . A p-value of <0.05 was considered statistically significant.

RESULTS

Table 1: Age Group Distribution of Children

		Frequency	Percent
Age Group	<1 Year	26	27.1%
	1 to 2 years	18	18.8%
	2 to 3 years	15	15.6%
	3 to 4 years	17	17.7%
	4 to 5 years	20	20.8%
	Total	96	100.0%

The study included a total of 96 children categorized into five age groups: under 1 year, 1 to 2 years, 2 to 3 years, 3 to 4 years, and 4 to 5 years. The highest proportion of children (27.1%) were aged under 1 year. The second highest percentage (20.8%) was observed in the 4 to 5 years age group. The lowest proportion (15.6%) was found in the 2 to 3 years age group.

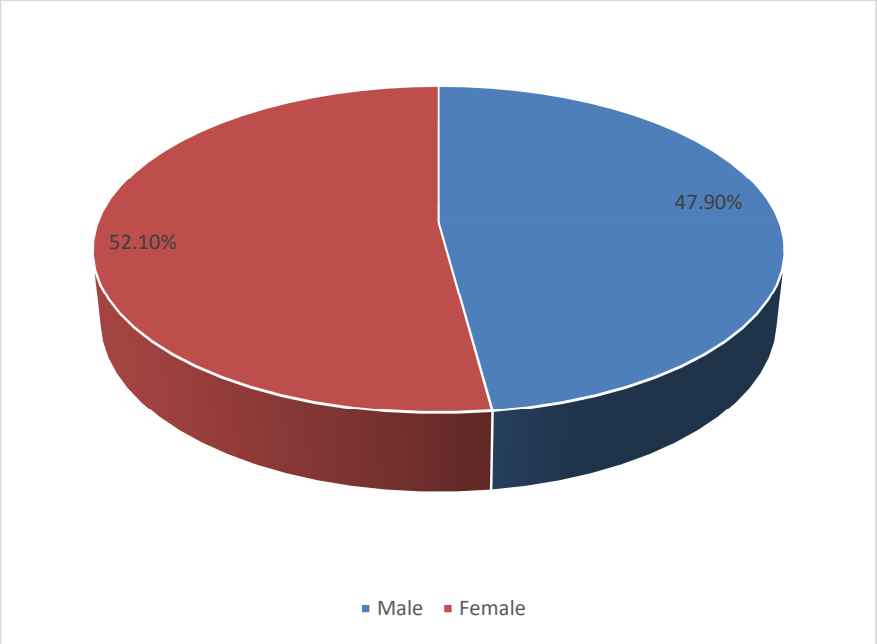


Graph 1: Age Group Distribution of Children

Table 2: Gender Distribution of Children

		Frequency	Percent
Gender	Male	46	47.9%
	Female	50	52.1%
	Total	96	100.0%

Out of the total 96 children included in the study, 46 (47.9%) were male and 50 (52.1%) were female.

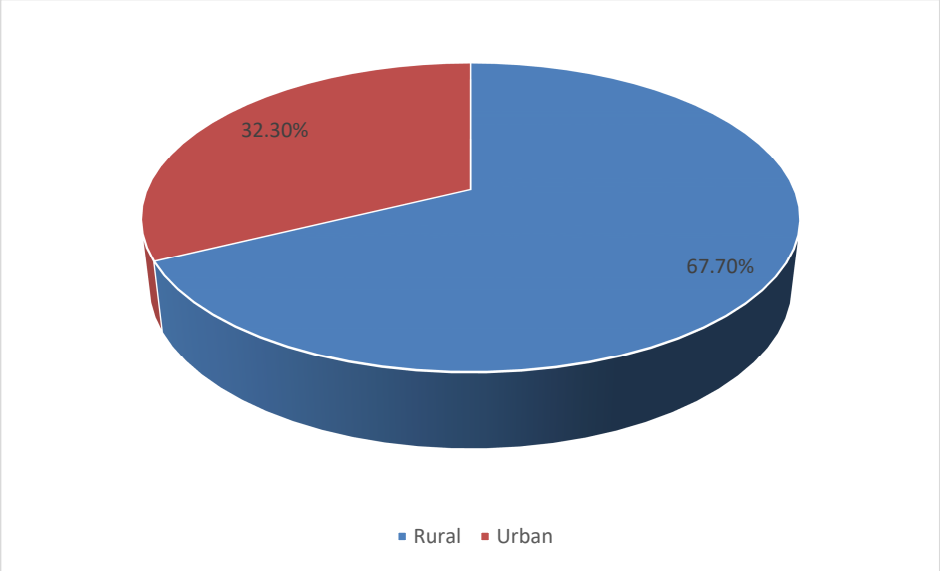


Graph2: Gender Distribution of Children

Table 3: Locality of residence Distribution of Children

		Frequency	Percent
Locality	Rural	65	67.7%
	Urban	31	32.3%
	Total	96	100.0%

Among the 96 children included in the study, 65 (67.7%) were from rural areas, while 31 (32.3%) were from urban areas.

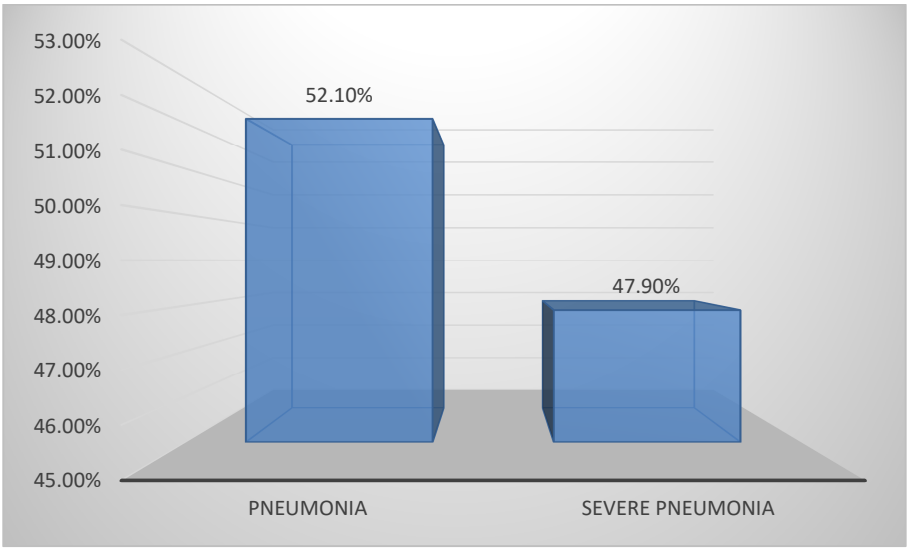


Graph 3: Locality Distribution of Children

Table 4 : Grade of Pneumonia Among Children as per revised WHO ARI classification 2014

		Frequency	Percent
Grade of Pneumonia	Pneumonia	50	52.1%
	Severe Pneumonia	46	47.9%
	Total	96	100.0%

Out of the 96 children included in the study, 50 (52.1%) were diagnosed with pneumonia, while 46 (47.9%) had severe pneumonia.

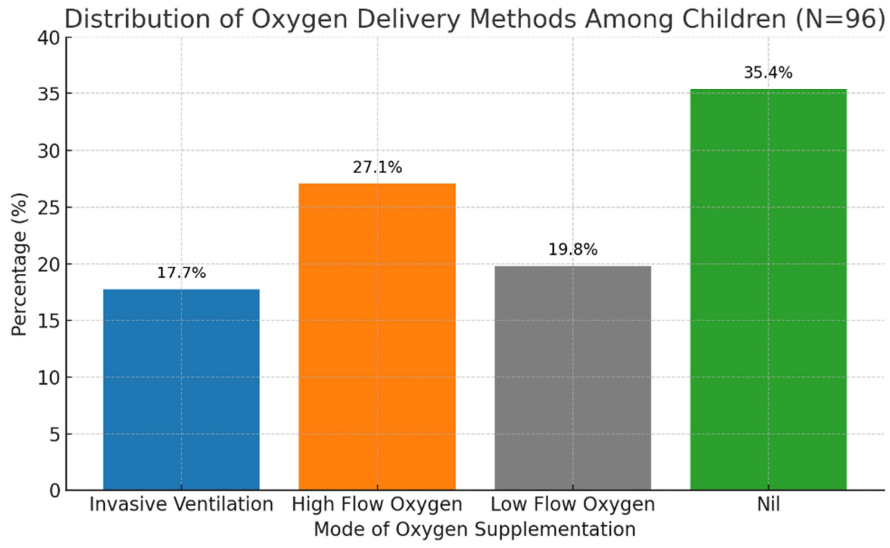


Graph 4: Grade of Pneumonia Among Children

Table 5: Mode of Oxygen delivery and Ventilation

		Frequency	Percent
Mode of Oxygen Supplementation	Invasive (Mechanical) Ventilation	17	17.7%
	High Flow Oxygen Device	26	27.08%
	Low Flow Oxygen Device	19	19.8%
	Nil	34	35.4%
	Total	96	100.0%

Among 96 chilldren included in study, 17 (17.7%) required invasive (mechanical ventilation) ventilation, while 26 (27%%) were managed with High Flow Oxygen delivery system and 19 (19.8%) received low flow oxygen delivery. A significant proportion of chilldren (34; 35.4%) did not require any form of oxygen support.

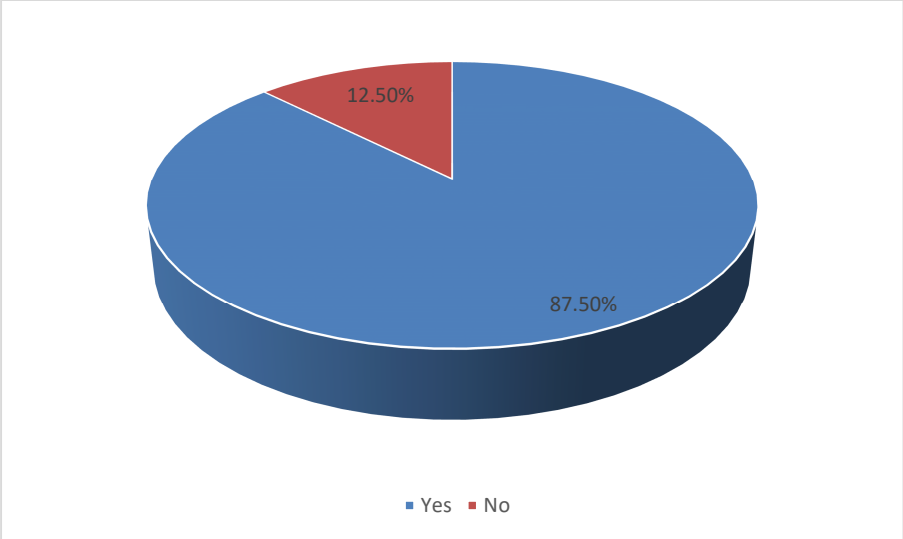


Graph 5: Mode of Oxygen delivery and mode of Ventilation

Table 6: National Immunization Schedule (NIS) Status

		Frequency	Percent
NIS- Vaccination Status	Yes	84	87.5%
	No	12	12.5%
	Total	96	100.0%

Among the 96 children included in the study, 84 (87.5%) had received vaccinations under the National Immunization Schedule (NIS), while 12 (12.5%) had not been vaccinated.

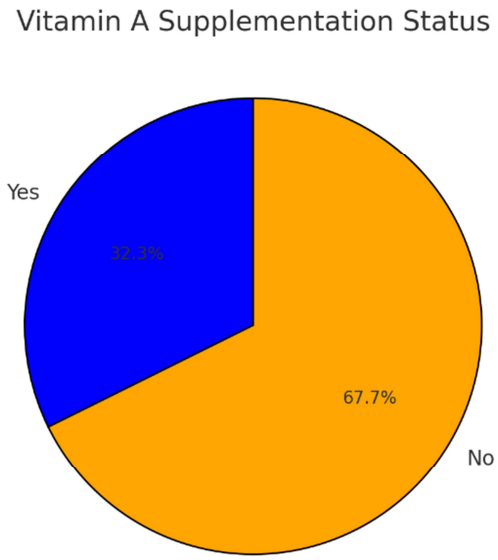


Graph 6: National Immunization Schedule (NIS) Status

Table 7: Vitamin A Supplementation Status

		Frequency	Percent
VIT A Supplementation – 1 dose at 9 month of age	Yes	31	32.29%
	No	65	67.7%
	Total	96	100.0%

Out of the 96 children included in the study, 31 (32.2%) had received vitamin A supplementation, while 65 (67.7%) had not.



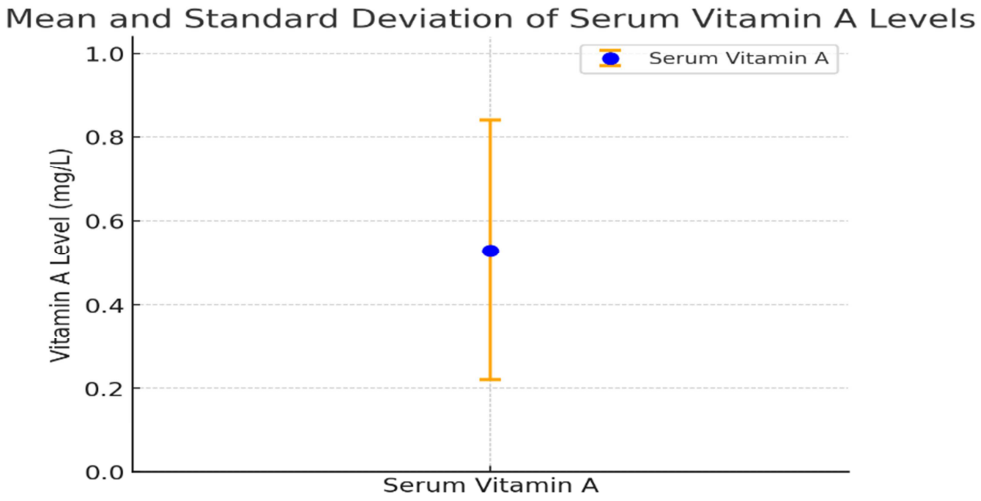
Graph 7: Vitamin A Supplementation Status

Table 8: Mean and Standard Deviation of Serum Vitamin A Levels

	Mean	Std. Deviation
Serum Vitamin A	0.53	0.31

Serum Vitamin A level >0.70 micromol/ L is considered normal, 0.70-0.35 micromol/ L is subclinical deficiency and <0.35 micromol/L is severe deficiency.

The mean serum vitamin A level among the 96 children included in the study was 0.53 with a standard deviation of 0.31.

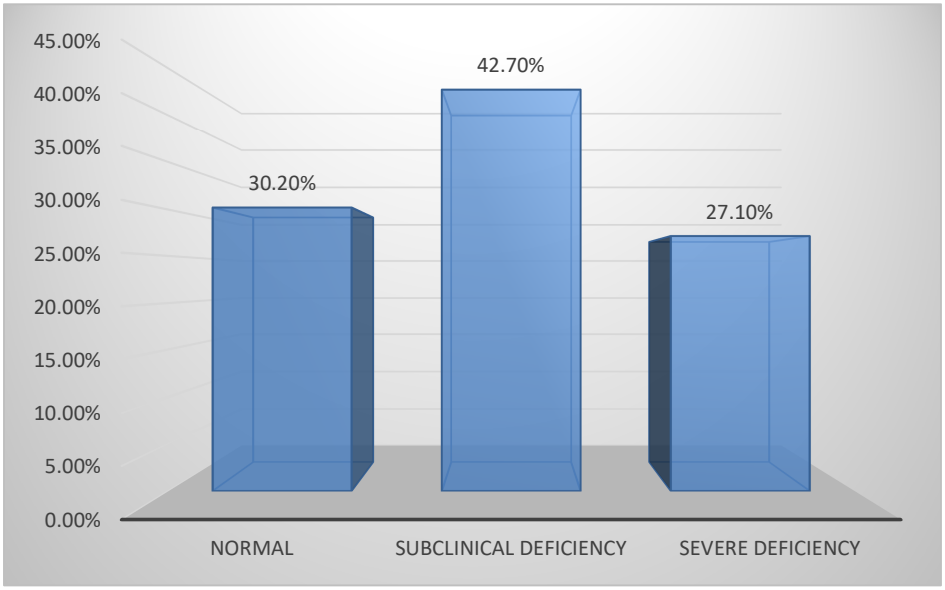


Graph 8 – Mean and Standard deviation of Serum Vitamin A level

Table 9: Vitamin A Status of Children

		Frequency	Percent
Vitamin A Status	Normal	29	30.2%
	Subclinical deficiency	41	42.7%
	Severe deficiency	26	27.1%
	Total	96	100.0%

Out of the 96 children included in the study, 29 (30.2%) had normal vitamin A levels, while 41 (42.7%) had subclinical vitamin A deficiency. A significant proportion of children (26; 27.1%) had severe vitamin A deficiency. The categorisation is based on WHO criteria.

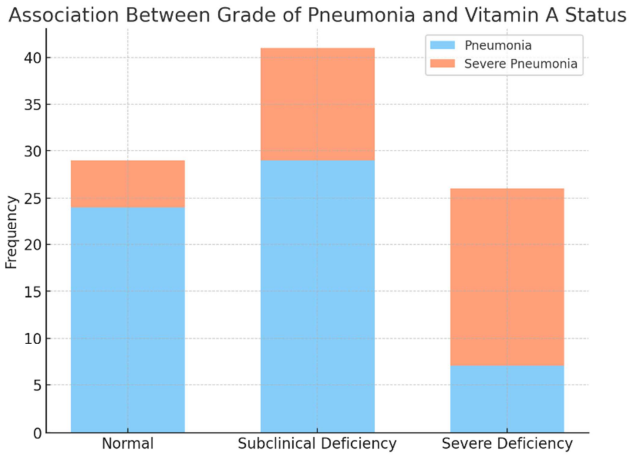


Graph 9: Vitamin A Status of Children

Table 10: Association Between Grade of Pneumonia and Vitamin A Status

Grade of Pneumonia				
Vitamin A Status		Frequency	Percent	P value
Normal	Pneumonia	24	82.7%	0.02
	Severe Pneumonia	5	17.2%	
	Total	29	100.0%	
Subclinical deficiency	Pneumonia	29	70.7%	
	Severe Pneumonia	12	29.3%	
	Total	41	100.0%	
Severe deficiency	Pneumonia	7	26.9%	
	Severe Pneumonia	19	73.1%	
	Total	26	100.0%	

Among children with normal vitamin A levels (n = 29), 82.7% had pneumonia, while 17.2% had severe pneumonia. Children with subclinical vitamin A deficiency (n = 41) showed a different pattern, with 70.7% having pneumonia and only 29.3% having severe pneumonia. Among those with severe deficiency, Only 26.9% had pneumonia, but a striking 73.1% had severe pneumonia. The p-value for this association was 0.02, indicating a statistically significant relationship between vitamin A status and the severity of pneumonia.

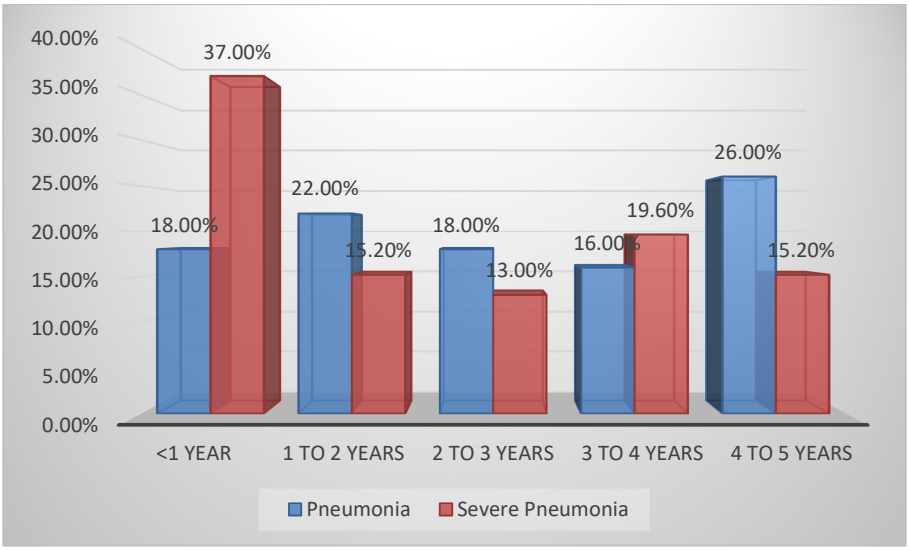


Graph 10 – Association between grade of Pneumonia and Vitamin A status

Table 11: Association Between Grade of Pneumonia and Age Group

			Grade of pneumonia		Total	P value
			Pneumonia	Severe Pneumonia		
Age Group	<1 year	n	9	17	26	0.06
		%	18.0%	37.0%	27.1%	
	1 to 2 years	n	11	7	18	0.55
		%	22.0%	15.2%	18.8%	
	2 to 3 years	n	9	6	15	0.69
		%	18.0%	13.0%	15.6%	
	3 to 4 years	n	8	9	17	0.84
		%	16.0%	19.6%	17.7%	
	4 to 5 years	n	13	7	20	0.29
		%	26.0%	15.2%	20.8%	
Total		n	50	46	96	0.22
		%	100.0%	100.0%	100.0%	

Among children under 1 year (n = 26), 18.0% had pneumonia, while 37.0% had severe pneumonia. In the 1 to 2 years age group (n = 18), 22.0% had pneumonia, while only 15.2% had severe pneumonia. In the 2 to 3 years age group (n = 15), 18.0% had pneumonia, and 13.0% had severe pneumonia. Similarly, in the 3 to 4 years age group (n = 17), 16.0% had pneumonia, and 19.6% had severe pneumonia. Among children aged 4 to 5 years (n = 20), 26.0% had pneumonia, while 15.2% had severe pneumonia. The overall p-value for the association was 0.22, indicating that the relationship between age group and grade of pneumonia is not statistically significant.

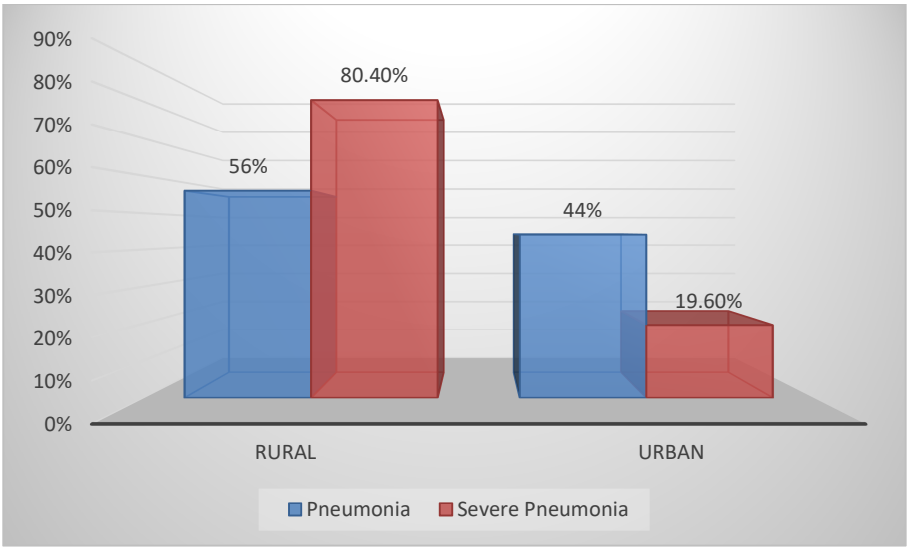


Graph 11: Association Between Grade of Pneumonia and Age Group

Table 12: Association Between Grade of Pneumonia and Locality

			Grade of pneumonia		Total	P value
			Pneumonia	Severe Pneumonia		
Locality	Rural	n	28	37	65	0.01
		%	43.07%	56.92%	67.7%	
	Urban	n	22	9	31	
		%	70.9%	29.03%	32.29%	
Total		n	50	46	96	
		%	52.08%	47.91%	100.0%	

Among the 65 children from rural areas, 43.07% had pneumonia, while 56.9% had severe pneumonia. Among the 31 children from urban areas, 70.9% had pneumonia, and only 29% had severe pneumonia. The p-value for this association was 0.01, indicating that the relationship between pneumonia severity and locality is statistically significant.

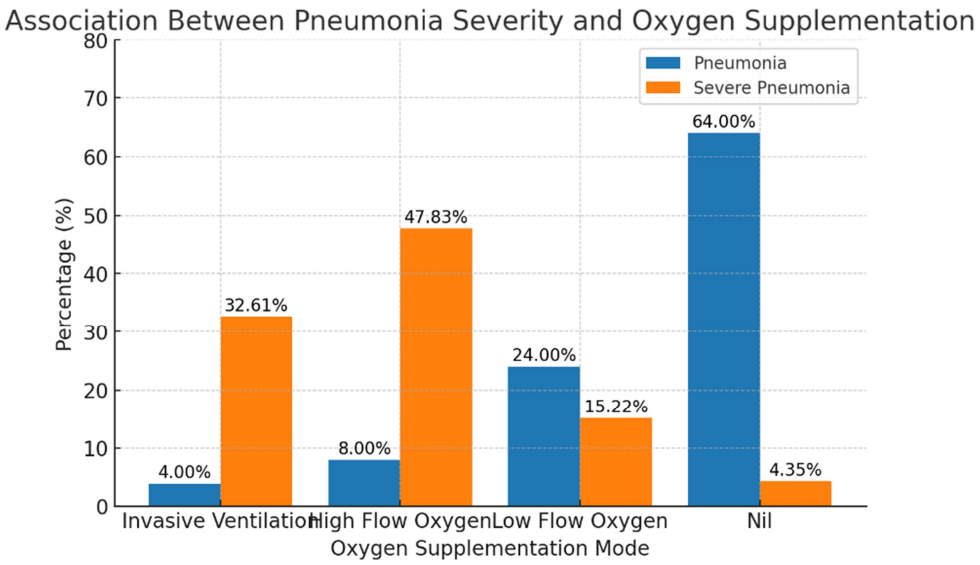


Graph 12: Association Between Grade of Pneumonia and Locality

Table 13: Association Between Grade of Pneumonia and mode of Oxygen Supplementation

			Grade of Pneumonia			
			Pneumonia	Severe Pneumonia	Total	p Value
Oxygen Supplementation	Invasive Ventilation	n	2	15	17	
		%	4.00%	32.61%	17.71%	0.001
	High Flow Oxygen Device	n	4	22	26	
		%	8.00%	47.83%	27.08%	0.001
	Low Flow Oxygen Device	N	12	7	19	
		%	24.00%	15.22%	19.79%	0.002
	Nil	N	32	2	34	
		%	64.00%	4.35%	35.42%	0.001
	Total	N	50	46	96	
		%	100.00%	100.00%	100.00%	0.001
	As % of Total cases		52.08%	47.92%	100.00%	

This table shows the association between the grade of pneumonia and the mode of oxygen supplementation**.** 4% of children with pneumonia required Invasive Ventilation, 8% required High-flow Oxygen, 24% required Low-flow Oxygen, and 64% did not require Oxygen. Among children with Severe Pneumonia**,** 32.61% required Invasive Ventilation, 47.83% required High-flow Oxygen, 15.22% required Low-flow Oxygen, and 4.35% did not require oxygen. The overall **p-value (<0.001)** indicates a statistically significant association between Pneumonia severity and Oxygen requirement, showing that Severe Pneumonia cases required more intensive respiratory support.

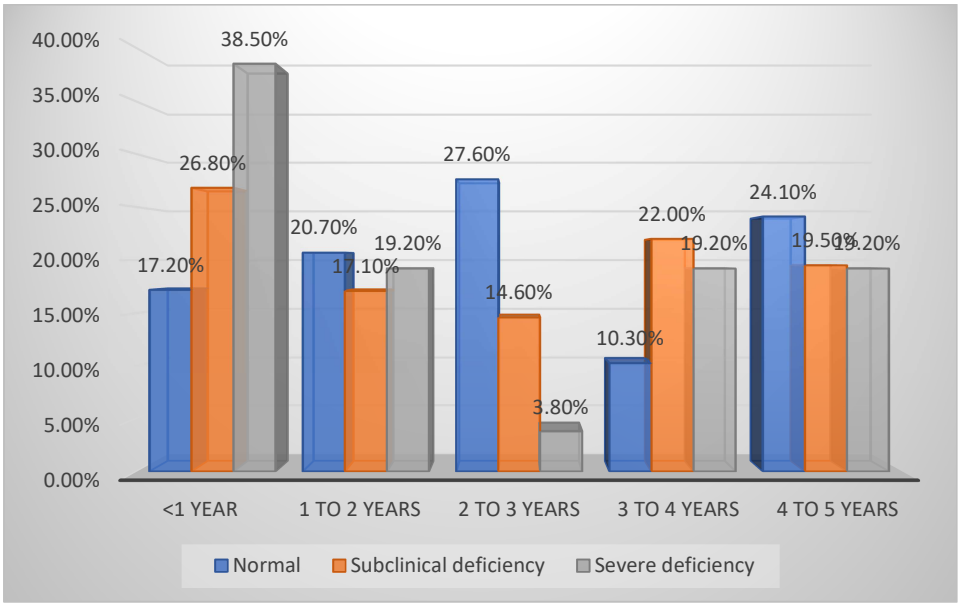


Graph 13: Association Between Grade of Pneumonia and Oxygen Supplementation

Table 14: Association Between Vitamin A Status and Age Group

			Vitamin A Status			Total	P Value
			Normal	Subclinical deficiency	Severe deficiency		
Age Group	<1 Year	N	5	11	10	26	0.33
		%	17.2%	26.8%	38.5%	27.1%	
	1 to 2 years	N	6	7	5	18	0.97
		%	20.7%	17.1%	19.2%	18.8%	
	2 to 3 years	N	8	6	1	15	0.12
		%	27.6%	14.6%	3.8%	15.6%	
	3 to 4 years	N	3	9	5	17	0.63
		%	10.3%	22.0%	19.2%	17.7%	
	4 to 5 years	N	7	8	5	20	0.97
		%	24.1%	19.5%	19.2%	20.8%	
Total		N	29	41	26	96	0.34
		%	100.0%	100.0%	100.0%	100.0%	

Among children under 1 year, 38.5% had severe vitamin A deficiency. In the 1 to 2 years age group, 19.2% had severe deficiency, while the 2 to 3 years group had the lowest rate of severe deficiency at 3.8%. In the 3 to 4 years age group, 19.2% of children had severe deficiency, and in the 4 to 5 years group, the rate was 19.2% as well. Subclinical deficiency was most common among children under 1 year (26.8%) and between 3 to 4 years (22.0%). The p-value for the association between vitamin A status and age group was 0.34, indicating that the association is not statistically significant.

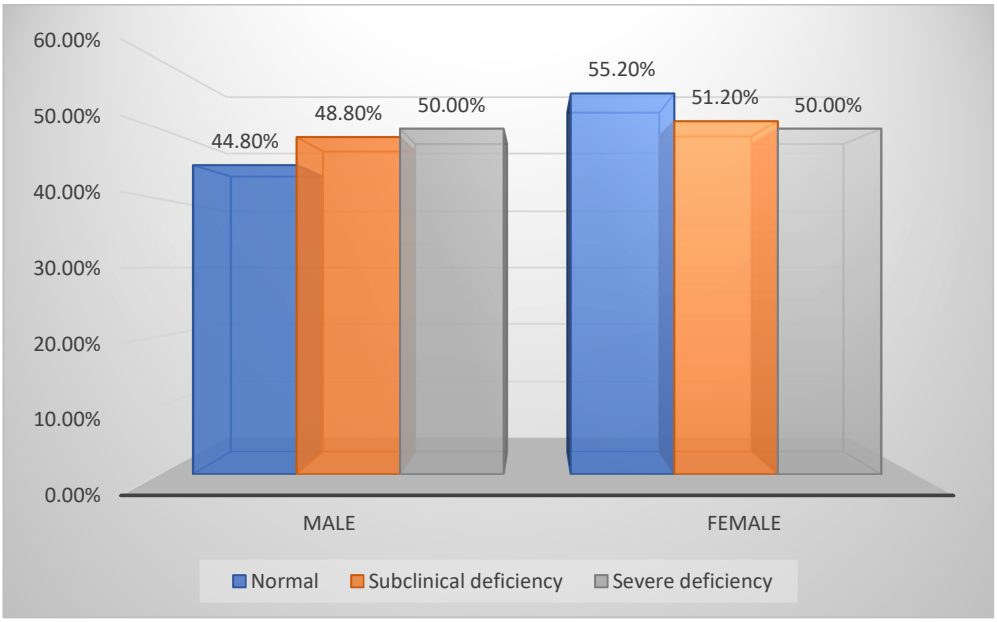


Graph 14: Association Between Vitamin A Status and Age Group

Table 15: Association Between Vitamin A Status and Gender

			Vitamin A Status			Total	P Value
			Normal	Subclinical deficiency	Severe deficiency		
Gender	Male	n	13	20	13	46	0.91
		%	44.8%	48.8%	50.0%	47.9%	
	Female	n	16	21	13	50	
		%	55.2%	51.2%	50.0%	52.1%	
Total		n	29	41	26	96	
		%	100.0%	100.0%	100.0%	100.0%	

Among the 46 male children, 44.8% had normal vitamin A levels, 48.8% had subclinical deficiency, and 50.0% had severe deficiency. Similarly, among the 50 female children, 55.2% had normal vitamin A levels, 51.2% had subclinical deficiency, and 50.0% had severe deficiency. Subclinical deficiency was more in females, and severe deficiency was similar frequency in both females and males. The p-value for this association was 0.91, indicating that the relationship between vitamin A status and gender is not statistically significant.

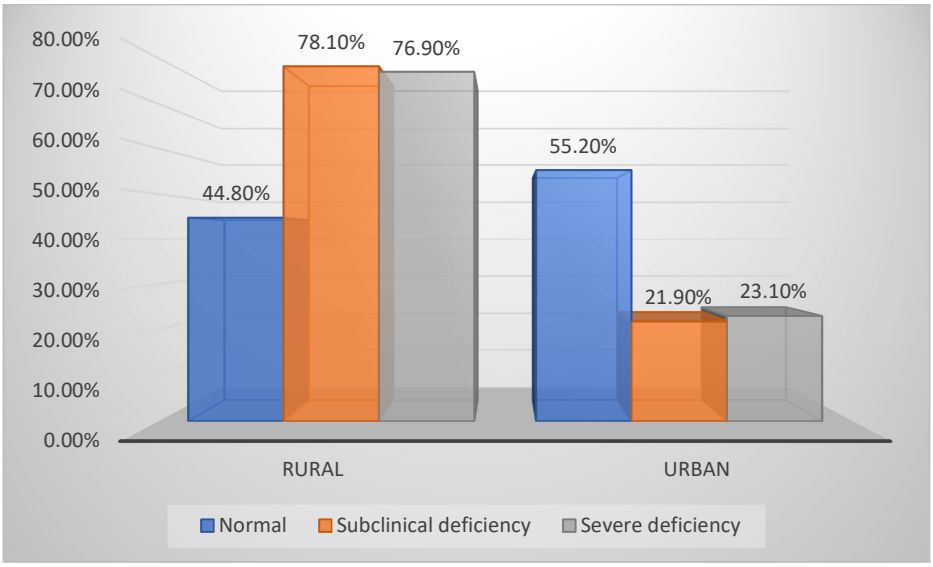


Graph 15: Association Between Vitamin A Status and Gender

Table 16: Association Between Vitamin A Status and Locality

			Vitamin A Status			Total	P value
			Normal	Subclinical deficiency	Severe deficiency		
Locality	Rural	n	13	32	20	65	0.01
		%	44.8%	78.1%	76.9%	67.7%	
	Urban	n	16	9	6	31	
		%	55.2%	21.9%	23.1%	32.3%	
Total		n	29	41	26	96	
		%	100.0%	100.0%	100.0%	100.0%	

Among the 65 children from rural areas, 44.8% had normal vitamin A levels, while a striking 78.1% had subclinical deficiency and 76.9% had severe deficiency. In contrast, among the 31 children from urban areas, 55.2% had normal vitamin A levels, while only 21.9% had subclinical deficiency and 23.1% had severe deficiency. The p-value for this association was 0.01, indicating a statistically significant relationship between vitamin A status and locality.



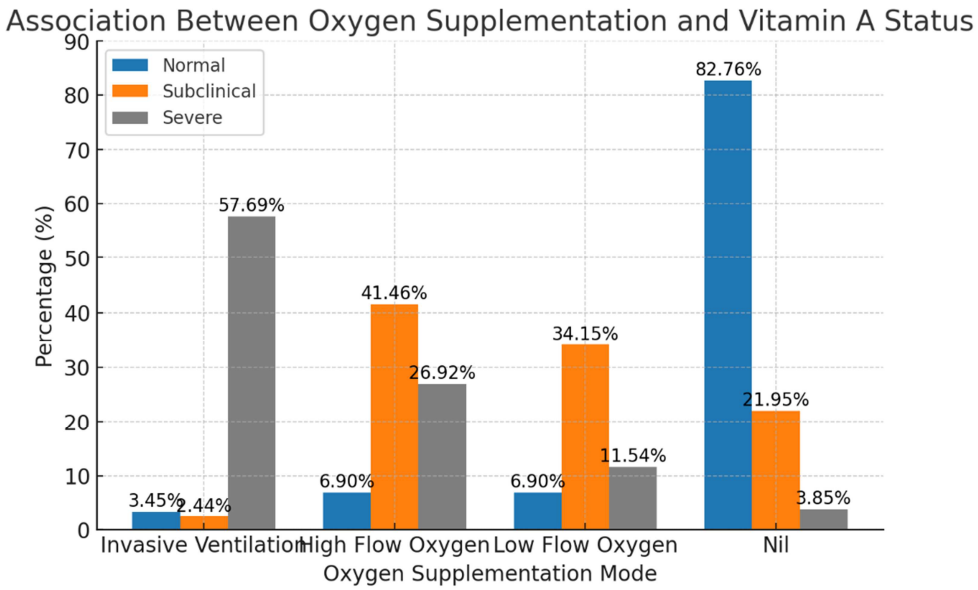
Graph 16: Association Between Vitamin A Status and Locality

Table 17 Association Between mode of Oxygen Supplementation and "Vitamin A" Status

			Vit A St atus				
			Normal	Sub clinical	Severe	Total	p Value
Oxygen Supplementation	Invasive Ventilation	n	1	1	15	17	0.001
		%	3.45%	2.44%	57.69%	17.71%	
	High Flow Oxygen Device	n	2	17	7	26	0.001
		%	6.90%	41.46%	26.92%	27.08%	
	Low Flow Oxygen Device	n	2	14	3	19	0.002
		%	6.90%	34.15%	11.54%	19.79%	
	Nil	n	24	9	1	34	0.001
		%	82.76%	21.95%	3.85%	35.42%	
	Total	n	29	41	26	96	0.001
		%	100.00%	100.00%	100.00%	100.00%	
	As % of Total cases		30.21%	42.71%	27.08%	100.00%	

This table shows the association between the mode of oxygen supplementation and Vitamin A status. 3.45% of children with normal Vitamin A status required invasive ventilation, 6.90% required high-flow oxygen, 6.90% required low-flow oxygen, and 82.76% did not require oxygen. Among children with severe Vitamin A deficiency, 57.69% required invasive ventilation, 26.92% required high-flow oxygen, 11.54% required low-flow oxygen, and 3.85% did not require oxygen. In children with subclinical Vitamin A deficiency, 2.44% required invasive ventilation, 41.46% required high-flow oxygen, 34.15% required low-flow oxygen, and 21.95% did not

require oxygen. The overall p-value (<0.001) indicates a highly significant association, showing that severe Vitamin A deficiency is linked to a higher need for invasive ventilation, subclinical deficiency is associated with higher use of non-invasive oxygen support, while children with normal Vitamin A levels mostly did not require oxygen therapy.

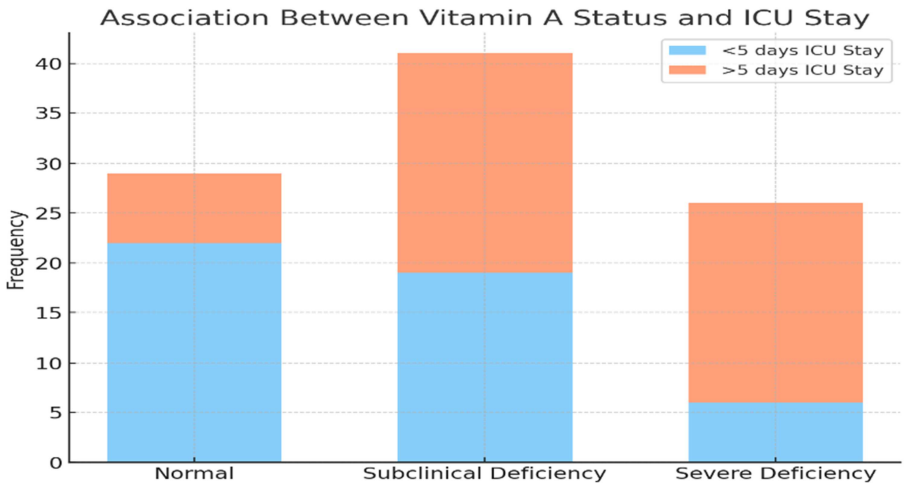


Graph 17: Association Between Oxygen Supplementation and "Vitamin A" Status

Table 18: Association Between Vitamin A Status and ICU Stay

	Normal	Subclinical Deficiency	Severe Deficiency	Total	P Value
<5 days	22	19	6	47	0.001
>5 days	7	22	20	49	
TOTAL	29	41	26	96	

Among the 47 children who stayed in the ICU for less than 5 days, 22 (46.8%) had normal vitamin A levels, 19 (40.4%) had subclinical deficiency, and 6 (12.76%) had severe deficiency. Among the 45 children who stayed in the ICU for more than 5 days, 7 (15.5%) had normal vitamin A levels, 22 (48.8%) had subclinical deficiency, and 20 (44.4%) had severe deficiency. The p-value for this association was 0.001, indicating that the relationship between vitamin A status and ICU stay duration is statistically significant.

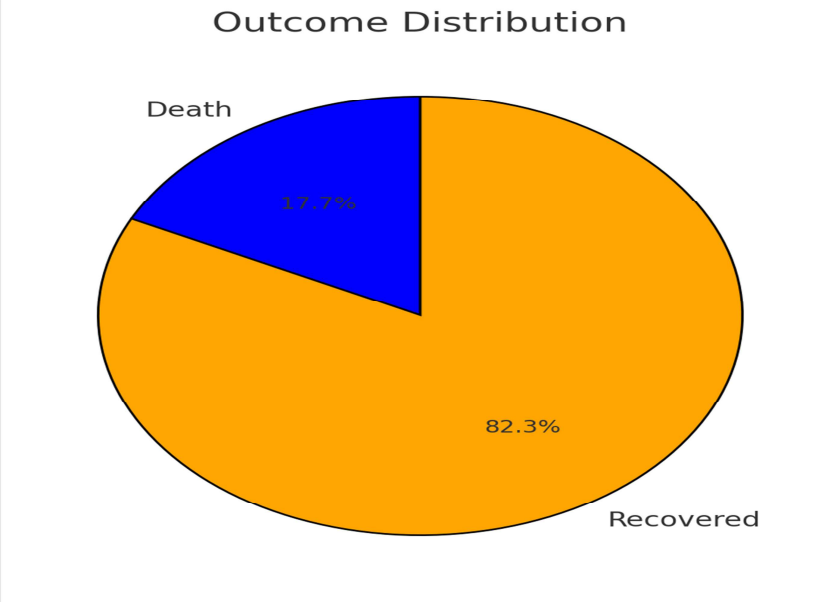


Graph 18 – Association between Vitamin A status and ICU stay

Table 19: Outcome of Children Based on Vitamin A Status

		Frequency	Percent
Outcome	Death	17	17.7%
	Recovered	79	82.2%
	Total	96	100.0%

Among the 96 children included in the study, 79 (82.2%) recovered, while 17 (17.7%) died.

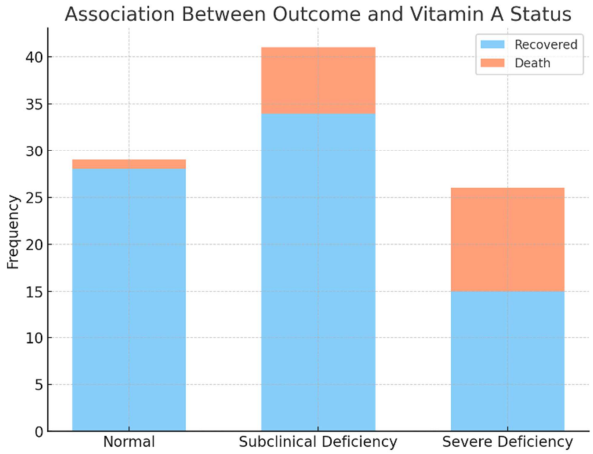


Graph 19: Outcome of Children Based on Vitamin A Status

Table 20: Association Between Outcome and Vitamin A Status

Outcome				
Vitamin A Status		Frequency	Per cent	P value
Normal	Death	1	3.4%	0.001
	Recovered	28	96.5%	
	Total	29	100.0%	
Subclinical deficiency	Death	7	17.07%	
	Recovered	34	82.9%	
	Total	41	100.0%	
Severe deficiency	Death	11	42.3%	
	Recovered	15	57.6%	
	Total	26	100.0%	

Among the 29 children with normal vitamin A levels, 1 (3.4%) died, while 28 (96.5%) recovered. Among the 41 children with subclinical vitamin A deficiency, 7 (17.07%) died, and 34 (82.9%) recovered. Among the 26 children with severe vitamin A deficiency, 11 (42.3%) died, while 15 (57.6%) recovered. The p-value for this association was 0.001, indicating that the relationship between vitamin A status and outcome is statistically significant.



Graph 20 – Association between Outcome and Vitamin A status

DISCUSSION

Vitamin A plays a crucial role in maintaining epithelial integrity, immune response, and overall lung health. This study aimed to evaluate the prevalence of vitamin A deficiency in children diagnosed with pneumonia and its association with the severity and clinical outcome of pneumonia. The findings reflect that vitamin A deficiency is highly prevalent among children with pneumonia, consistent with global evidence highlighting the link between poor vitamin A status and increased vulnerability to respiratory infections. Vitamin A deficiency impairs the immune system by reducing the production of protective antibodies, compromising the integrity of the respiratory epithelium, and increasing susceptibility to infection. Furthermore, low serum vitamin A levels are associated with delayed recovery, prolonged hospital stay, and higher rates of complications such as severe pneumonia and respiratory failure.

Age

In this study, highest proportion of children (27.1%) were aged under 1 year, indicating that infants are more vulnerable to health complications such as pneumonia and vitamin A deficiency. The second highest percentage (20.8%) was observed in the 4 to 5 years age group, suggesting that vitamin A deficiency and pneumonia persist even in older children. The lowest proportion (15.6%) was found in the 2 to 3 years age group. The relatively higher percentage of children under 1 year reflects their increased susceptibility to nutritional deficiencies and infections due to underdeveloped immunity and limited dietary intake. The balanced distribution across the other age groups highlights that vitamin A deficiency and pneumonia are not limited to infancy but continue to affect children well into early childhood. This

pattern suggests the need for continued nutritional and healthcare interventions beyond infancy to reduce the burden of vitamin A deficiency and related health complications.

Jiang et al., conducted a study on 88 children aged less than 3 years with pneumonia. The mean age of the study population was 8 ± 7 months. Among the children, 69 cases were under 1 year, and 19 cases were between 1 to 3 years. The study found no significant difference in serum vitamin A levels between different age groups ($p > 0.05$), but younger children had a higher prevalence of vitamin A deficiency, which was associated with increased risk of pneumonia severity and recurrent respiratory infections (RRI) within one year of discharge.[89]

Li et al., conducted a retrospective study on 181 children diagnosed with Mycoplasma pneumoniae pneumonia (MPP), with a median age of 46 months (IQR: 26.25–72.25 months) in the general MPP (GMPP) group and 55 months (IQR: 37.00–83.00 months) in the refractory MPP (RMPP) group. There was no significant difference in age between the two groups ($p = 0.062$).[90]

Abolurin et al conducted a study on 170 children aged 6 months to 59 months in South-Western Nigeria. The mean age of the study population was 16.8 ± 9.0 months. There was no statistically significant difference in the prevalence of vitamin A deficiency (VAD) between children aged ≤24 months and those aged >24 months ($p = 0.159$).[91]

Kundu et al analyzed data from the Comprehensive National Nutrition Survey (CNNS) conducted in India between 2016–18. The study included children aged 12 to 59 months. The highest prevalence of vitamin A deficiency (VAD) was observed in the 36–47 months age group (20.82%), while the lowest prevalence was reported in

the 18–23 months age group (12.47%). The prevalence of VAD increased with age, but the difference was not statistically significant ($p = 0.262$).[83]

Gultom et al. (2020) conducted a study on 594 children under the age of 5 years in West Java. Among the sample, 36% of children were younger than 3 years, while 64% were aged 3 to 5 years. The study found no significant association between age and pneumonia incidence ($p > 0.05$).[84]

Gender

In this study, Out of the total 96 children included in the study, 46 (47.9%) were male and 50 (52.1%) were female. The gender distribution was relatively balanced, with a slightly higher proportion of female participants. The balanced gender representation suggests that vitamin A deficiency and pneumonia are not significantly influenced by gender, affecting both male and female children almost equally. This finding indicates that nutritional deficiencies and susceptibility to infections like pneumonia are widespread across both genders.

Jiang et al., reported that out of the 88 children with pneumonia, 68 were male and 20 were female, giving a male-to-female ratio of 3.4:1. The serum vitamin A levels were 0.9 ± 0.3 μmol/L in males and 0.8 ± 0.3 μmol/L in females, but the difference was not statistically significant ($p > 0.05$), indicating that vitamin A status and pneumonia severity were not influenced by gender.[89]

Li et al reported that among the 181 children with MPP, 82 were male and 70 were female in the GMPP group, while 18 were male and 11 were female in the RMPP group. The male-to-female ratio was similar between the groups, and the difference was not statistically significant ($p = 0.422$).[90]

Abolurin et al reported that among the 170 children included in the study, 89 (52.4%) were male and 81 (47.6%) were female. The prevalence of VAD was not significantly different between male and female children ($p = 1.000$).[91]

Kundu et al reported that the prevalence of VAD was almost equal among male and female children. Among male children, the prevalence was 17.81%, whereas in female children, it was 17.23%. The difference between male and female prevalence was not statistically significant ($p = 0.082$).[83]

Gultom et al. (2020) reported that the sample included an almost equal distribution of boys and girls. However, the study did not find any significant difference in the incidence of pneumonia based on gender ($p > 0.05$).[84]

Locality

In this study, Among the 96 children included in the study 65 (67.7%) were from rural areas, while 31 (32.3%) were from urban areas. This indicates that a larger proportion of children affected by vitamin A deficiency and pneumonia were from rural backgrounds. The higher prevalence of cases in rural areas suggests that children in these areas may face greater challenges in terms of access to healthcare, nutritional resources, and overall living conditions. Limited access to vitamin A-rich foods, inadequate healthcare infrastructure, and poor sanitation in rural areas could contribute to higher rates of nutritional deficiencies and respiratory infections. The relatively lower percentage of urban cases (32.3%) may reflect better healthcare access, improved nutritional practices, and enhanced living standards in urban settings.

Abolurin et al. (2018) noted that 89 (59.4%) of the children were from a low social class, while 69 (40.6%) were from a high social class. The prevalence of VAD did not differ significantly between social classes ($p = 0.740$).[91]

Kundu et al found that the prevalence of VAD was slightly higher in rural areas (17.76%) compared to urban areas (16.86%). However, the difference was not statistically significant ($p = 0.262$).[83]

Oxygen Supplementation

In this study, among the 96 children, 17 (17.7%) required invasive ventilation, while 45 (46.9%) were managed with non-invasive oxygen therapy (high-flow or low-flow oxygen devices). A significant proportion of children (34; 35.4%) did not require any form of oxygen support. The relatively high percentage of children requiring non-invasive oxygen therapy (46.9%) reflects the severity of respiratory distress associated with pneumonia, particularly in cases of vitamin A deficiency. The fact that 17.7% of children required invasive ventilation indicates that a considerable number of cases progressed to a critical stage, necessitating intensive respiratory support. The need for oxygen supplementation, especially invasive ventilation, highlights the clinical severity of pneumonia and the potential role of compromised immunity due to malnutrition and vitamin A deficiency.

Li et al found that 45.39% of children with GMPP required oxygen supplementation compared to 100% of children with RMPP ($p < 0.001$). This suggests that children with refractory pneumonia had more severe respiratory distress, requiring higher levels of respiratory support.[90]

Vitamin A Status

In this study, Out of the 96 children included in the study, 29 (30.2%) had normal vitamin A levels, while 41 (42.7%) exhibited subclinical vitamin A deficiency. A significant proportion of children (26; 27.1%) had severe vitamin A deficiency. The mean serum vitamin A level among the 96 children included in the study was 0.53 with a standard deviation of 0.31. The high percentage of subclinical and severe vitamin A deficiency reflects a significant public health concern, as vitamin A plays a crucial role in immune function, growth, and overall health. Subclinical deficiency, though not immediately symptomatic, can weaken immunity and increase vulnerability to infections such as pneumonia. Severe vitamin A deficiency is more concerning, as it is linked to higher rates of morbidity and mortality due to its impact on immune function and epithelial integrity. This suggests that nearly 70% of the study population had some degree of vitamin A deficiency, highlighting the urgent need for improved nutritional programs and vitamin A supplementation.

Jiang et al. (2016) reported that among the 88 children, 35 (40%) had vitamin A deficiency (VAD), 25 (28%) had suspected subclinical vitamin A deficiency (SSVAD), and 28 (32%) had normal vitamin A levels. Severe pneumonia cases had a higher prevalence of VAD (63%) compared to mild pneumonia cases (28%), reinforcing the association between low vitamin A levels and increased severity of pneumonia.[89]

Li et al reported that the prevalence of vitamin A deficiency (VAD) was significantly higher in children with RMPP (68.75%) compared to those with GMPP (31.75%) ($p = 0.004$). Adjusted serum vitamin A levels were significantly lower in

the RMPP group (12.23 mg/L) than in the GMPP group (17.00 mg/L) and the control group (25.10 mg/L) ($p < 0.001$).[90]

Abolurin et al. (2018) reported that the mean serum retinol level among the children was 1.33 ± 0.38 μmol/L (range: 0.50–2.25 μmol/L). Nine children (5.3%) had VAD, but none had severe VAD. There was no statistically significant association between VAD and age, gender, social class, immunization status, or nutritional status.[91]

Kundu et al reported that the overall prevalence of vitamin A deficiency (VAD) among children aged 12–59 months in India was 17.54%. Children from poorer economic sections had higher VAD rates compared to those from richer sections. The prevalence of VAD was highest in children with stunting (18.24%) and severe stunting (21.72%) compared to non-stunted children (17.41%).[83]

Gultom et al. (2020) reported that 38.6% of children under five years of age did not receive vitamin A supplementation. The overall prevalence of vitamin A deficiency was associated with an increased risk of pneumonia; however, the association was not statistically significant (OR = 1.011; 95% CI 0.690–1.481; p = 1.000).[84]

Grade of Pneumonia and Vitamin A Status

In this study, Out of the 96 children included in the study, 50 (52.1%) were diagnosed with pneumonia, while 46 (47.9%) had severe pneumonia. In this study, association between the grade of pneumonia and vitamin A status among the 96 children included in the study shows significant differences in the severity of pneumonia based on vitamin A levels. Among children with normal vitamin A levels (n = 29), 48.3% had pneumonia, while 51.7% had severe pneumonia. This indicates

that even in children with normal vitamin A levels, the occurrence of severe pneumonia was relatively high. Children with subclinical vitamin A deficiency (n = 41) showed a different pattern, with 70.7% having pneumonia and only 29.3% having severe pneumonia. This suggests that subclinical deficiency is more strongly associated with milder forms of pneumonia. However, among children with severe vitamin A deficiency (n = 26), the trend was reversed. Only 26.9% had pneumonia, but a striking 73.1% had severe pneumonia. This indicates that severe vitamin A deficiency is closely linked to an increased risk of developing severe forms of pneumonia. These findings suggest that vitamin A plays a critical role in immune function and respiratory health. While subclinical deficiency may predispose children to milder pneumonia, severe deficiency significantly increases the risk of progression to more severe forms of pneumonia.

Jiang et al. (2016) divided the study population into two groups based on the severity of pneumonia: 58 children had mild pneumonia, and 30 children had severe pneumonia. The serum vitamin A levels were significantly lower in the severe pneumonia group (0.7 ± 0.3 μmol/L) compared to the mild pneumonia group (0.9 ± 0.3 μmol/L) ($p < 0.05$). The detection rate of vitamin A deficiency was also higher in the severe pneumonia group (63%) compared to the mild pneumonia group (28%) ($p < 0.05$), suggesting that low vitamin A levels are associated with increased severity of pneumonia.[89]

Li et al. (2020) demonstrated that adjusted vitamin A levels were significantly lower in children with severe pneumonia (RMPP) (12.23 mg/L) compared to those with mild pneumonia (GMPP) (17.00 mg/L) ($p < 0.001$). Multivariate logistic regression analysis showed that low vitamin A levels were independently associated

with increased odds of developing RMPP (OR = 0.795, 95% CI: 0.669–0.946; p = 0.010).[90]

Vitamin A Status and Oxygen Supplementation

In this study, the association between oxygen supplementation and vitamin A status among the 96 children included in the study shows varying patterns based on the severity of vitamin A deficiency. Among children with normal vitamin A levels (n = 29), 3.45% required invasive ventilation, 6.90% required high-flow oxygen, 6.90% required low-flow oxygen, and 82.76% did not require any form of oxygen supplementation. Children with subclinical vitamin A deficiency (n = 41) showed a lower need for invasive ventilation, with only 2.44% requiring it. However, 41.46% required high-flow oxygen, 34.15% required low-flow oxygen, and 21.95% did not require oxygen supplementation. In contrast, among children with severe vitamin A deficiency (n = 26), 57.69% required invasive ventilation, which was the highest among the groups. Similarly, 26.92% of children with severe deficiency required high-flow oxygen, 11.54% required low-flow oxygen, while only 3.85% did not need oxygen support. The higher need for invasive ventilation among children with severe vitamin A deficiency suggests a potential link between deficiency and increased severity of respiratory distress. The findings suggest that while mild and subclinical deficiency may increase the risk of milder respiratory complications, severe deficiency may contribute to more severe respiratory failure, necessitating intensive respiratory support.

Li et al reported that vitamin A deficiency was more prevalent in children requiring oxygen supplementation. Among children with RMPP, all cases (100%) required oxygen supplementation, and these children had significantly lower vitamin A levels compared to those with GMPP ($p < 0.001$).[90]

Grade of Pneumonia and Oxygen Supplementation

In this study, association between the grade of pneumonia and the need for oxygen supplementation among the 96 children included in the study shows a clear and statistically significant pattern. Among the 13 children who required invasive ventilation, only 2.0% had pneumonia, but a striking 26.1% had severe pneumonia. This indicates that invasive ventilation was primarily needed for severe cases of pneumonia. Among the 44 children who required non-invasive ventilation, 24.0% had pneumonia, while 69.6% had severe pneumonia. This suggests that non-invasive ventilation was more commonly used in severe cases, although it was also required in some milder cases. In contrast, among the 39 children who did not require oxygen supplementation, 74.0% had pneumonia, and only 4.3% had severe pneumonia. This shows that most cases of mild pneumonia were managed without the need for oxygen support. These findings suggest that severe pneumonia cases are more likely to require intensive respiratory support, including invasive and non-invasive ventilation. The significantly higher need for ventilation in severe cases reflects the clinical severity and respiratory compromise associated with pneumonia, particularly in children with nutritional deficiencies and weakened immunity.

Li et al found that children with RMPP had a significantly higher need for oxygen supplementation compared to those with GMPP (100% vs. 45.39%; $p < 0.001$). This indicates that severe pneumonia cases are more likely to require respiratory support.[90]

Vitamin A Status and Duration of ICU Stay

In this study, association between vitamin A status and duration of ICU stay among the 96 children included in the study shows varying patterns based on the

severity of vitamin A deficiency. Among the 47 children who stayed in the ICU for less than 5 days, 22 (46.8%) had normal vitamin A levels, 19 (40.4%) had subclinical deficiency, and 6 (12.7%) had severe deficiency. This indicates that shorter ICU stays were more common among children with normal or subclinical vitamin A levels. Among the 49 children who stayed in the ICU for more than 5 days, 7 (14.2%) had normal vitamin A levels, 22 (44.8%) had subclinical deficiency, and 20(40.8%) had severe deficiency. The higher proportion of children with severe vitamin A deficiency in this group suggests that severe deficiency may be linked to prolonged ICU stays. Severe vitamin A deficiency may weaken immune response and increase the severity of illness, leading to prolonged hospitalization.

Li et al found a significant negative correlation between adjusted vitamin A levels and length of stay (LOS) in the hospital ($r = -0.384$; $p < 0.001$). Children with lower vitamin A levels had longer hospital stays, indicating that vitamin A deficiency may contribute to prolonged recovery from pneumonia.[90]

Outcome and Vitamin A Status

In this study, Among the 96 children included in the study, 79 (82.2%) recovered, while 17 (17.7%) died. The high recovery rate (82.2%) reflects the effectiveness of medical interventions and supportive care provided to the children. However, the 17.7% mortality rate is concerning and indicates that a significant proportion of children with pneumonia and vitamin A deficiency experienced severe clinical outcomes.

In this study, association between clinical outcomes and vitamin A status among the 96 children included in the study shows varying patterns based on the severity of vitamin A deficiency. Among the 29 children with normal vitamin A

levels, 1(3.4%) died, while 28 (96.5%) recovered. This suggests that even among children with adequate vitamin A levels, there was mortality, possibly due to the severity of pneumonia or other underlying health conditions. In contrast, among the 41 children with subclinical vitamin A deficiency, 7 (17.07%) died, and 34 (82.9%) recovered. This indicates that subclinical deficiency was associated with better overall survival rates compared to severe deficiency groups. Among the 26 children with severe vitamin A deficiency, 11 (42.3%) died, while 15 (57.6%) recovered. The higher mortality rate in the severe deficiency group suggests that severe vitamin A deficiency may weaken immune response and reduce the body's ability to fight infections, increasing the risk of poor clinical outcomes.

Jiang et al. (2016) followed up the children for one year after discharge and found that 82 cases fully recovered, while 6 cases showed clinical improvement. Among children with vitamin A deficiency, the incidence of recurrent respiratory infection (RRI) within one year was 49%, significantly higher than the incidence among children with normal vitamin A levels (18%) ($p < 0.05$). The results suggest that vitamin A deficiency increases the risk of recurrent respiratory infections after pneumonia.[89]

Li et al. (2020) reported that vitamin A deficiency was linked to poor clinical outcomes. Children with RMPP and low vitamin A levels had a significantly longer duration of fever (10 days vs. 5 days; $p < 0.001$) and longer hospital stays (9 days vs. 6 days; $p < 0.001$). This suggests that low vitamin A levels are associated with more severe clinical outcomes and prolonged recovery.[90]

Abolurin et al reported that the overall prevalence of VAD was 5.3%, which is lower than previous studies conducted in Nigeria. The study concluded that the

reduction in VAD prevalence may be attributed to improved immunization coverage and vitamin A supplementation programs.[91]

Kundu et al reported that vitamin A deficiency was associated with poor nutritional outcomes. The prevalence of VAD was higher in children with anemia (20.66%) compared to non-anemic children (16.10%) ($p = 0.000$). Similarly, the prevalence of VAD was higher in severely stunted children (21.72%) than non-stunted children (17.36%) ($p = 0.001$).[83]

Gultom et al found that 26.8% of children under five years of age suffered from pneumonia. The incidence of pneumonia was higher among children who did not receive vitamin A supplementation, but the difference was not statistically significant ($p = 1.000$).[84]

RECOMMENDATIONS

Based on the findings, recommendations can be made to address the burden of vitamin A deficiency and its impact on pneumonia severity in children. First, there is a need to strengthen vitamin A supplementation programs, particularly in rural areas where deficiency rates and pneumonia severity were highest. Expanding outreach and improving supply chains for vitamin A supplementation could help close the gap in coverage and improve immune health among children. Second, improving nutritional education for caregivers and healthcare providers is essential to promote dietary diversity and increase the intake of vitamin A-rich foods, such as green leafy vegetables, dairy products, and fortified foods. Third, targeted public health interventions, including regular screening for vitamin A levels and early identification of at-risk children, could help prevent the progression of subclinical deficiency to severe deficiency and its associated complications. Finally, improving immunization coverage and addressing gaps in vaccination could further reduce the burden of respiratory infections and improve child health outcomes.

STRENGHTS OF STUDY

This is a first of its kind study to estimate prevalence of Vitamin A deficiency in North Karnataka. Previous studies have been conducted in other countries and in Northern parts of India. This study also assesses the correlation between Vitamin A status and other factors like severity of pneumonia, duration of ICU stay, and outcome. Adequate sample size has been analysed.

LIMITATIONS OF STUDY

The limitations of the study are that it was a single centric study and cannot be generalized to a larger population.

CONCLUSION

The cross sectional observational study highlights a significant burden of Vitamin A deficiency among children with Pneumonia, with 42.7% of children exhibiting subclinical deficiency and 27.1% having severe deficiency. Severe Vitamin A deficiency was strongly associated with increased “Severe Pneumonia”, higher need for Invasive Ventilation, and even mortality in 42.3 %. Subclinical deficiency had higher prevalence of 42.7% and was associated with increasing number of cases with “Pneumonia”. Children from rural areas had significantly higher rates of deficiency and severe pneumonia compared to urban children. Although the overall recovery rate was 82.2%, mortality and duration of ICU stay was higher among children with severe vitamin A deficiency.

SUMMARY

This study examined the association between vitamin A deficiency and pneumonia severity among children aged 2 months to 5 years. The study population included 96 children, with the highest proportion of cases observed in the under 1 year age group (27.1%). The gender distribution was balanced, with 47.9% male and 52.1% female participants. A higher proportion of cases were from rural areas (67.7%) compared to urban areas (32.3%), and vitamin A deficiency was significantly more common among rural children ($p = 0.01$), reflecting disparities in healthcare access and nutrition.

Regarding vitamin A status, 30.2% of children had normal vitamin A levels, 42.7% had subclinical deficiency, and 27.1% had severe deficiency. Severe deficiency was most prevalent among children under 1 year (38.5%), although the association between vitamin A status and age group was not statistically significant ($p = 0.34$). The mean serum vitamin A level was 0.53 with a standard deviation of 0.31, indicating low overall vitamin A levels among the study population.

Pneumonia severity was strongly linked to vitamin A status. Severe pneumonia was more common among children with severe vitamin A deficiency (73.1%) compared to those with normal levels (51.7%). The association between vitamin A status and pneumonia severity was statistically significant ($p = 0.02$). The need for oxygen supplementation was higher among children with severe deficiency; 46.1% of severely deficient children required invasive ventilation, while 46.1% needed non-invasive ventilation. The association between vitamin A status and oxygen supplementation was statistically significant ($p = 0.002$).

Vitamin A supplementation was reported in 32.2% of children, but 67.7% had not received supplementation, reflecting a gap in public health outreach. The study showed that severe vitamin A deficiency was linked to prolonged ICU stay and poorer clinical outcomes. Among children with severe deficiency, 42.3 % died, compared to 17.07% in the subclinical group and 3.4% in the normal group. The association between vitamin A status and mortality was statistically significant ($p = 0.001$).

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ANNEXURE – I - INFORMED CONSENT FORM

"VITAMIN A STATUS IN CHILDREN WITH PNEUMONIA; A HOSPITAL BASED, CROSS SECTIONAL STUDY"

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

Introduction: The purpose of the proposed study is to measure the level of serum vitamin A in children diagnosed with Pneumonia. This is being done in order to establish a connection between severity and frequency of pneumonia and vitamin A deficiency. The results of the study will help guide appropriate treatment measures.

Explanation of procedure: Upon consent, peripheral venous sample will be drawn from the patient at the time of admission, and processed to measure serum vitamin A. All costs shall be borne by the investigator. Parents will be informed the report of the test upon processing.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: benefits by participating in this study- the parents will receive a report of the child's vitamin A level so the child can be treated for deficiency if present and necessary. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator. Authorization for publication of aggregated data:** Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact:. If you have any question or complaints with regard to your right as study participant you may contact Dr.Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am, on behalf of my child, making a voluntary decision to let my child participate in the study **"VITAMIN A STATUS IN CHILDREN WITH PNEUMONIA; A HOSPITAL BASED, CROSS SECTIONAL STUDY"**. My signature below indicates that I have, on behalf of my child, decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator

Signature of the investigator

ANNEXURE – II - PROFORMA

NAME OF THE PATIENT : IP NUMBER :

AGE / SEX

ADDRESS

RESIDENTIAL STATUS- URBAN/ RURAL

MEDICAL HISTORY- CHIEF COMPLAINTS

PAST HISTORY

FAMILY HISTORY

VITAMIN A SUPPLEMENTATION – YES / NO

IMMUNIZATION STATUS: COMPLETE /INCOMPLETE

AREA OF RESIDENCE: RURAL / URBAN

GENERAL PHYSICAL EXAMINATION -

VITALS - HR-

RR-

SPO2-

TEMP-

BP-

HEAD TO TOE EXAMINATION -

ANTHROPOMETRY-

HEIGHT OBSERVED	HEIGHT EXPECTED	CENTILE /SCORE
WEIGHT - OBSERVED	WEIGHT EXPECTED	CENTILE/SCORE
MID-ARM CIRCUMFERENCE OBSERVED	MUAC EXPECTED	CENTILE /SCORE
WEIGHT FOR HEIGHT		CENTILE/SCORE

7)SYSTEMIC EXAMINATION

RESPIRATORY SYSTEM- UPPER RESPIRATORY TRACT- NOSE

OROPHARYNX

SINUSES

LOWER RESPIRATORY TRACT

INSPECTION - SHAPE OF CHEST

MOVEMENT WITH RESPIRATION

WORK OF BREATHING

RESPIRATORY RATE

PALPATION- CONFIRMATION OF RESPIRATORY MOVEMENTS

PERCUSSION- IDENTICAL AREAS ON BITH SIDES

AUSCULTATION- INTENSITY OF BREATH SOUNDS

TYPE OF BREATH SOUNDS

ADVENTITIOUS SOUNDS

GRADING OF PNEUMONIA-

TACHYPNEA	+ / -	GRADE
LOWER CHEST INDRAWING	+/-	GRADE
DANGER SIGNS	+/-	GRADE

CVS-

PER ABDOMEN-

CNS -

INVESTIGATIONS-

HB	
WBC	
N/L/E/M	
HSCRP	
CHEST XRAY	
BLOOD C/S	
ABG	
OTHER TESTS	

9)DIAGNOSIS -

10)PLAN OF TREATMENT -

11) COURSE OF ILLNESS-

ANTIBIOTIC USED	
DAYS OF ANTIBIOTIC	
O2 SUPPLEMENTATION	YES/NO HIGH FLOW O2 DEVICE LOW FLOW O2 DEVICE
MECHANICAL VENTILATION	
DURATION OF ICU STAY	< 5 DAYS >5 DAYS

SERUM VITAMIN A -

>0.70 micromol/ L	Normal
<0.70 micromol/L	Subclinical deficiency
<0.35 micromol/L	Severe Deficiency

ANNEXURE – III –

MASTER CHART

Timestamp	AGE (YRS)	SEX	RR (bpm)	SPO2	HEIGHT CENTILE	WEIGHT CENTILE	GRADE OF PNEUMONIA - TACHYPNEA	GRADE OF PNEUMONIA- GRUNT/RETRACTIONS/DANGER SIGNS	GRADE OF PNEUMONIA	HB	WBC	HSCRP	BLOOD C/S	DIAGNOSIS	ANTIBIOTICS USED	OXYGEN SUPPLEMENTATION	SERUM VITAMIN A	VITAMIN A STATUS	STATUS	AREA OF RESIDENCE	NIS VACCINATION COMPLETE?	VIT A SUPPLEMENTATION
6-3-2024 23:14:29	4	MALE	38	92			PRESENT	ABSENT	PNEUMONIA	10.9	5.2	15	Nogc	Urti with age	Amox	NASAL PRONGS/ HOOD	0.4286	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
6-3-2024 23:15:46	4	MALE	45	88			PRESENT	PRESENT	SEVERE PNEUMONIA	9.2	15.3	148	Nogc	ARDS	Piptaz, amika, linid, vanco	CPAP/ HFNC	0.46	SUBCLINICAL DEFICIENCY	DEATH	U	Y	N
6-3-2024 23:17:15	2.5	FEMALE	48	93			PRESENT	ABSENT	PNEUMONIA	9	8.4	0.9	Nogc	Bronchilitis	Clarithromycin, amoxiclav		0.75	NORMAL	RECOVERED	U	Y	N
6-3-2024 23:18:34	4	MALE	40	96			PRESENT	ABSENT	PNEUMONIA	11.3	10.8	0.2	Nogc	Bronchilitis	Amoxiclav		0.393	SUBCLINICAL DEFICIENCY	RECOVERED	R	N	Y
6-3-2024 23:21:27	2	MALE	60	88			PRESENT	PRESENT	SEVERE PNEUMONIA	8	14.5	94	Nogc	Down syndrome w lrti	Piptaz, amika	NASAL PRONGS/ HOOD	0.28	SEVERE DEFICIENCY	DEATH	R	Y	N
6-3-2024 23:24:16	7 MONTHS	MALE	60	888	0	-2	PRESENT	PRESENT	SEVERE PNEUMONIA	10.5	3.9	100	STAPH EPIDERMIDIS	LEFT UPPER LOBE PNEUMONIA WITH SEVERE DEHYDRATION WITH STAPH SEPSIS	PIPTAZ, AMIKA	CPAP/ HFNC	0.426	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	Y
6-3-2024 23:26:40	5	MALE	32	95	0	0	PRESENT		PNEUMONIA	9.4	5.5	185	NOGC, EAR STAPH AUREUS	LRTI	AMOXICLAV	NO	0.2857	SEVERE DEFICIENCY	DEATH	U	Y	Y
6-3-2024 23:28:18	2.6	MALE	35	95	-2 TO -3	-2	PRESENT	ABSENT	PNEUMONIA	18.3	9.6	2.8	NOGC	BRONCHIOLITIS	AMOXICLAV	NO	0.46	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	N
6-3-2024 23:30:00	1.2	FEMALE	60	88	0	0 TO -1	PRESENT	PRESENT	SEVERE PNEUMONIA	11.1	14.1	70.9	NOGC	BRONCHOPNEUMONIA	PIPTAZ, AMIKA	CPAP/ HFNC	0.4286	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
6-3-2024 23:31:31	4	MALE	40	95	0 TO +1	-1 TO 0	PRESENT	PRESENT	SEVERE PNEUMONIA	11.4	9.1	33.3	NOGC	BRONCHIOLITIS	AMOXICLAV	NASAL PRONGS/ HOOD	0.321	SEVERE DEFICIENCY	RECOVERED	R	Y	N
6-3-2024 23:33:24	3.1	MALE	35	95	0	-1	PRESENT	ABSENT	PNEUMONIA	11.9	11.6	1.8		LRTI	AMOXICLAV	NO	1.07	NORMAL	RECOVERED	R	N	Y
6-3-2024 23:34:39	1.5	MALE	40	95	+1	+1	PRESENT	ABSENT	PNEUMONIA	13	11.8	18	NOGC	LRTI	CLARITHROMYCIN	NO	0.3339	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
6-3-2024 23:37:48	3.2	MALE	68	82	<-3	<-3	PRESENT	PRESENT	SEVERE PNEUMONIA	9.2	9.4	177.2	STAPH PYOGENES	SAM WITH BRONCHOPNEUMONIA WITH RIGHT PLEURAL EFFUSION IN RESPIRATORY FAILURE	LINID, VANCO, CLINDA	MECHANIAL VENTILATION	0.3214	SEVERE DEFICIENCY	DEATH	R	Y	N
6-3-2024 23:40:18	5	FEMALE	35	96	-1	-1	PRESENT	ABSENT	PNEUMONIA	13	9.2	4		LRTI	AMOXICLAV	NO	0.42	SUBCLINICAL DEFICIENCY	RECOVERED	R	N	N
6-3-2024 23:42:07	1.5	MALE	45	92	0 TO -1	0	PRESENT	ABSENT	PNEUMONIA	15	11	20		RT LOWER LOBE PNEUMONIA	AMOXICLAV	NASAL PRONGS/ HOOD	0.46	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N
6-3-2024 23:43:53	4.6	MALE	40	94	0	0 TO -1	PRESENT	ABSENT	PNEUMONIA	8.2	19.6	12.8	NOGC	ACUTE GE WITH LRTI	XONE	NO	0.4143	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	N
6-3-2024 23:46:08	0.3	MALE	68	86	<-3	<-3	PRESENT	PRESENT	SEVERE PNEUMONIA	15	15	44	KLEBSIELLA PNEUMONIA	ASPIRATION PNEUMONIA WITH KLEBSIELLA SEPSIS	PIPTAZ, VANCO, FLUCONAZOLE, LINID	CPAP/ HFNC	0.19	SEVERE DEFICIENCY	RECOVERED	R	N	N
6-3-2024 23:47:50	1	FEMALE	55	93	+2	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	8.8	11.8	120		BRONCHOPNEUMONIA	AMOXICLAV, AMIKA	NASAL PRONGS/ HOOD	0.285	SEVERE DEFICIENCY	RECOVERED	U	Y	Y
6-3-2024 23:49:56	0.2	MALE	74	90	-2	-2	PRESENT	PRESENT	SEVERE PNEUMONIA	16	26	66	CANDIDA GLABRATA	S/P TEF REPAIR, RT MIDDLE LOBE CONSOLIDATION, CANDIDA SEPSIS	PIPTAZ, MEROPENEM, AMIKA	CPAP/ HFNC	0.1714	SEVERE DEFICIENCY	RECOVERED	U	Y	Y
6-3-2024 23:52:08	4.5	FEMALE	48	84	+1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	10.4	6.1	270	ACINETOBACTER BAUMANII	ARDS WITH SEPSIS	MEROPENEM , AMIKA	MECHANIAL VENTILATION	0.1179	SEVERE DEFICIENCY	DEATH	R	Y	N
6-3-2024 23:53:57	0.4	FEMALE	60	92	-2	-3	PRESENT	PRESENT	SEVERE PNEUMONIA	15.4	16.9	94		RT LOWER LOBE CONSOLIDATION	PIPTAZ, AMIKA	CPAP/ HFNC	0.142	SEVERE DEFICIENCY	RECOVERED	R	Y	Y
6-3-2024 23:55:46	0.8	MALE	50	95	+1	0	PRESENT	ABSENT	PNEUMONIA	10.1	7.1	1.3	NOGC	BRONCHIOLITIS, PUSTUKE ON ARM	CLARITHROMYCIN	NO	0.5	SUBCLINICAL DEFICIENCY	RECOVERED	R	N	N
6-3-2024 23:57:55	3.5	FEMALE	60	84	0	-1	PRESENT	PRESENT	SEVERE PNEUMONIA	11.8	13.2	98	NOGC	ARDS WITH FEBRILE SEIZURE	PIPTAZ, AMIKA, CLINDA	MECHANIAL VENTILATION	0.25	SEVERE DEFICIENCY	RECOVERED	R	Y	Y
6-3-2024 23:59:45	1	FEMALE	50	94	-2 TO -3	-3	PRESENT	ABSENT	PNEUMONIA	8.8	11.8	42	NOGC	SAM WITH LRTI	AMOXICLAV	NO	0.285	SEVERE DEFICIENCY	RECOVERED	R	Y	Y
6-4-2024 0:01:29	0.7	MALE	48	93	-3	-3	PRESENT	PRESENT	SEVERE PNEUMONIA	8.3	3.7	267		RT LOWER LOBE PNEUMONIA	PIPTAZ, AMIKA	NO	0.278	SEVERE DEFICIENCY	RECOVERED	R	N	N
6-4-2024 0:03:46	0.5	FEMALE	68	90	<-3	<-3	PRESENT	PRESENT	SEVERE PNEUMONIA	15	20.3	25	NOGC	BRONCHIOLITIS	AMOXICLAV	CPAP/ HFNC	0.178	SEVERE DEFICIENCY	RECOVERED	R	Y	N
6-4-2024 0:05:28	4.5	MALE	40	94	-1	-2	PRESENT	ABSENT	PNEUMONIA	8.2	19.6	12.8	NOGC	AGE WITH LRTI	XONE, AMIKA	NO	0.1143	SEVERE DEFICIENCY	RECOVERED	U	Y	Y
6-4-2024 0:07:14	0.3	FEMALE	58	94	-2	-2	PRESENT	ABSENT	PNEUMONIA	10.4	3.2	1.4	STAPH AUREUS	RT EAR ASOM WITH LRTI	AMOXICLAV	NO	0.339	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N

6-4-2024 0:09:07	1.1	MALE	32	95	-1	-2	PRESENT	ABSENT	PNEUMONIA	9.2	15.8	3.5	NOGC	AGE WITH SOME DEHYDRATION, WITH LRTI	XONE, AMIKA	NO	0.85	NORMAL	RECOVERED	R	Y	Y
6-4-2024 0:10:50	1.1	FEMALE	40	95	+1	+1	PRESENT	ABSENT	PNEUMONIA	10.9	12.4	14.4	NOGC	LT EAR ASOM WITH LRTI	CIPLOX	NO	1.0714	NORMAL	RECOVERED	R	N	Y
6-4-2024 0:12:35	0.7	MALE	50	94	0	-3 TO -2	PRESENT	PRESENT	PNEUMONIA	14.4	9.1	63		BRONCHOPNEUMONIA	AMOXICLAV	NASAL PRONGS/ HOOD	0.107	SEVERE DEFICIENCY	RECOVERED	R	N	Y
6-4-2024 0:14:23	0.9	FEMALE	45	96	-2	-3	PRESENT	ABSENT	PNEUMONIA	10.4	3.2	1.4	NOGC	LRTI WITH GERD, S/P TEF REPAIR	CLARITHROMYCIN	NO	0.6071	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	N
6-4-2024 0:16:05	0.7	MALE	80	88	-2	-3	PRESENT	PRESENT	SEVERE PNEUMONIA	10.6	10.7	13.6	NOGC	BRONCHOPNEUMONIA	CIPLOX, AMIKA, MERO	NASAL PRONGS/ HOOD	0.339	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	Y
6-4-2024 0:18:00	5	FEMALE	39	98	0	+2 TO +3	PRESENT	ABSENT	PNEUMONIA	8.1	19.8	251	NOGC	SDNS WITH LRTI	AMOXICLAV	NO	0.5	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	Y
6-4-2024 0:21:46	5	FEMALE	38	94			PRESENT	ABSENT	PNEUMONIA	10.8	6.8			LRTI	AMOXICLAV	NO	0.1143	SEVERE DEFICIENCY	RECOVERED	U	Y	N
6-4-2024 0:23:28	0.2	FEMALE	68	92			PRESENT	PRESENT	PNEUMONIA	14.2	9.9	1.6		BRONCHIOLITIS	AMOXICLAV, AMIKA	NASAL PRONGS/ HOOD	0.6071	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N
6-4-2024 0:24:58	2.5	MALE	30	95			PRESENT	ABSENT	PNEUMONIA	13.8	5.6	12		LRTI	AMOXICLAV	NO	0.7857	NORMAL	RECOVERED	R	Y	Y
6-4-2024 0:26:47	0.4	MALE	55	86			PRESENT	PRESENT	SEVERE PNEUMONIA	10	11.8	10.4		K/C/O KRABBES DISEASE WITH LEFT LOWER LOBE PNEUMONIA	TAXIM, AMIKA	CPAP/ HFNC	0.66071	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
6-4-2024 0:28:46	4.5	MALE	30	95			PRESENT	ABSENT	PNEUMONIA	13	9.6	0.14		LRTI	AMOXICLAV	NO	0.42	SUBCLINICAL DEFICIENCY	RECOVERED	R	N	Y
6-4-2024 0:30:09	2	MALE	38	95			PRESENT	ABSENT	PNEUMONIA	10.1	18.2	12.6	NOGC	ACUTE GE WITH LRTI	XONE, AMIKA	NO	0.1179	SEVERE DEFICIENCY	RECOVERED	R	N	N
6-4-2024 13:19:47	5	FEMALE	35	95			PRESENT	ABSENT	SEVERE PNEUMONIA					LRTI		NO	1.017	NORMAL	RECOVERED	R	Y	N
6-4-2024 13:20:59	3	MALE	40	96			PRESENT	ABSENT	PNEUMONIA					BRONCHOPNEUMONIA		NO	0.37	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	Y
6-4-2024 13:23:22	2.5	FEMALE	40	92			PRESENT	ABSENT	PNEUMONIA	12	6.8	5		BRONCHOPNEUMONIA	AMOXICLAV, AMIKACIN	NASAL PRONGS/ HOOD	0.38	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N
6-4-2024 13:28:52	4	FEMALE	36	95			PRESENT	ABSENT	PNEUMONIA	12	11.6	15	NOGC	BRONCHOPNEUMONIA	AMOXICLAV	NO	0.53	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N
6-4-2024 13:31:20	2	FEMALE	32	98			PRESENT	ABSENT	PNEUMONIA	13	13.5	26		LEFT MIDDLE LOBE PNEUMONIA	PIPTAZ, AMIKA	NASAL PRONGS/ HOOD	0.4643	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	Y
6-4-2024 13:34:24	0.2	MALE	60	88			PRESENT	PRESENT	SEVERE PNEUMONIA	11.1	12.8	5	STAPH EPIDERMIDIS	RIGHT LOWER LOBE CONSOLIDAITON WITH STAPH AUREUS SEPSIS	MEROPENEM, VANCOMYCIN	CPAP/ HFNC	0.713	NORMAL	RECOVERED	R	Y	Y
6-4-2024 13:35:44	0.5	FEMALE	60	88			PRESENT	ABSENT	SEVERE PNEUMONIA	14.2	13.4	6		BRONCHOPNEUMONIA	CIPOX, MEROPENEM, AMIKACIN	CPAP/ HFNC	0.125	SEVERE DEFICIENCY	RECOVERED	R	Y	N
6-4-2024 13:49:36	0.3	FEMALE	60	93			PRESENT	PRESENT	SEVERE PNEUMONIA	10.3	23.2	95	KLEBSIELLA PNEUMONIA	RIGHT LOWER LOBE PNEUMONIA WITH KELBSIELLA SEPSIS	PIPTAZ, VANCOMYCIN, CLARITHROMYCIN	MECHANIAL VENTILATION	0.117	SEVERE DEFICIENCY	RECOVERED	R	N	Y
6-4-2024 13:51:57	0.8	FEMALE	62	93	0 TO +2	>+2	PRESENT	ABSENT	PNEUMONIA	8	51.4	130	ACINETOBACTER IWOFII	PNEUMONIA WITH ACINETOBACTER SEPSIS IN MODS	PIPTAZ, GENTA, MEROPENEM	MECHANIAL VENTILATION	0.85	NORMAL	DEATH	R	Y	N
6-4-2024 13:55:58	0.6	FEMALE	50	78	0 TO +2	0 TO -2	PRESENT	PRESENT	SEVERE PNEUMONIA	10.2	4.6	302	NOGC	BL PNEUMONIA WITH RT PLEURAL EFFUSION IN RDS WITH SEPSIS WITH PANCYTOPENIA ?ADENOVIRUS IN MODS IN SHOCK	CIPLOX, PIPTAZ, AMIKA, MEROPENEM	CPAP/ HFNC	0.145	SEVERE DEFICIENCY	DEATH	R	Y	N
11-3-2024 19:43:13	4	MALE	40	88	+2sd	0sd	PRESENT	PRESENT	SEVERE PNEUMONIA	10	18k	48	Nogc	Bronchopneumonia	Meropenem, vancomycin	CPAP/ HFNC	0.5	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	Y
11-3-2024 19:45:26	3	FEMALE	30	97	+1sd	+1sd	PRESENT	ABSENT	PNEUMONIA	12	9	15	Nogc	Viral pneumonia	amoxiclav	NO	0.75	NORMAL	RECOVERED	U	N	Y
11-5-2024 19:22:13	5	MALE	30	98	+2 sd	+1sd	PRESENT	ABSENT	PNEUMONIA	13	15	20	Nogc	Pneumonia	Amoxiclav	NO	1.07	NORMAL	RECOVERED	U	N	Y
11-5-2024 19:43:36	3.5	FEMALE	50	90	0	0	PRESENT	PRESENT	SEVERE PNEUMONIA	12	18	56	Nogc		Aspiration pneumonia	CPAP/ HFNC	0.28	SEVERE DEFICIENCY	RECOVERED	R	Y	Y
11-5-2024 19:45:17	2.4	MALE	22	93	0	0	PRESENT	ABSENT	PNEUMONIA	15	14	50	Nogc	Aspiration pneumonia	Piptaz, amikacin	NASAL PRONGS/ HOOD	0.42	SUBCLINICAL DEFICIENCY	RECOVERED	R	N	Y
11-5-2024 19:47:51	3.6	MALE	40	94	+1	+2	PRESENT	PRESENT	SEVERE PNEUMONIA	13	22	76	Acinetobacter baumanii	Acinetobacter baumanii sepsis with bronchopneumonia	Meropenem, vancomycin	CPAP/ HFNC	1.07	NORMAL	RECOVERED	R	Y	Y
11-5-2024 19:49:46	0.9	MALE	60	88	-1	0	PRESENT	PRESENT	SEVERE PNEUMONIA	13	23	75	Nogc	Bronchiolitis	Amoxicillin	CPAP/ HFNC	0.607	NORMAL	RECOVERED	U	Y	Y
11-5-2024 19:53:11	0.5	FEMALE	45	82	-1	-2	PRESENT	PRESENT	SEVERE PNEUMONIA	9.8	8	90	Candida kruzi	Candida kruzi sepsis with	Meropenem, vancomycin, fluconazole	CPAP/ HFNC	0.17	SEVERE DEFICIENCY	DEATH	R	Y	N
11-5-2024 19:55:03	0.7	MALE	38	98	+1	+1	PRESENT	ABSENT	PNEUMONIA	14	12	45	Nogc	Bronchiolitis	Amoxiclav	NASAL PRONGS/ HOOD	0.42	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	Y
11-5-2024 20:01:34	0.9	FEMALE	36	96	+1	+2	PRESENT	ABSENT	PNEUMONIA	13	6	56	Nogc	Bronchiolitis	Amoxicillin	NASAL PRONGS/ HOOD	0.33	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
11-5-2024 20:15:06	1.2	MALE	40	94	+1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	13	20	89	Nogc	Right lung Empyema with collapse	Meropenem, vancomycin, fluconazole	CPAP/ HFNC	0.78	NORMAL	RECOVERED	U	Y	Y
11-6-2024 23:44:12	3.5	MALE	30	95	-1	-2	PRESENT	ABSENT	PNEUMONIA	13	18	59	Nogc	UTI and LRTI	Piptaz, amikacin		0.42	NORMAL	RECOVERED	R	Y	Y
11-6-2024 23:46:38	0.8	FEMALE	70	83	-1	0	PRESENT	PRESENT	SEVERE PNEUMONIA	10	9	80		Adenoviral encephalitis with ARDS	Meropenem, vancomycin, oseltamivir, clarithromycin	MECHANIAL VENTILATION	0.33	SUBCLINICAL DEFICIENCY	DEATH	R	Y	N
11-6-2024 23:48:57	1.5	FEMALE	10	65	+1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	12	18	80	Enterobacter faecium sps	Enterobacter sepsis with Right side pleural effusion	Teicoplanin, metronidazole	MECHANIAL VENTILATION	1.07	NORMAL	DEATH	U	Y	N

11-6-2024 23:50:45	2.2	MALE	30	96	+1 to,+2	+1 to+2	PRESENT	ABSENT	PNEUMONIA	11	15	40	Nogc	Bronchiolitis	Clarithromycin	NASAL PRONGS/ HOOD	0.85	NORMAL	RECOVERED	R	N	Y
11-8-2024 17:30:10	4.8	MALE	45	95	-1	-2	PRESENT	PRESENT	SEVERE PNEUMONIA	13	15	40	Staphylococcus epidermidis	Staphylococcus epidermidis sepsis with bronchopneumonia	Linezolid, levofloxacin	MECHANIAL VENTILATION	1.07	NORMAL	DEATH	R	N	Y
11-8-2024 17:43:47	5	FEMALE	28	94	+2	+3	PRESENT	ABSENT	PNEUMONIA	13	16	25	Nogc	Bronchopneumonia	Amoxiciav	NO	0.857	NORMAL	RECOVERED	R	N	N
11-14-2024 23:33:18	4.7	MALE	36	93	-1	-1	PRESENT	PRESENT	SEVERE PNEUMONIA	12	14	35	Nogc	Bronchopneumonia	Piptaz, amikacin	CPAP/ HFNC	0.339	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N
11-15-2024 12:17:23	3.8	MALE	45	87	-2	-2	ABSENT	PRESENT	SEVERE PNEUMONIA	10	18	67	Burkholderia cepacia	Burkholderia sepsis with ARDS	Tigecycline, vancomycin	MECHANIAL VENTILATION	0.1143	SEVERE DEFICIENCY	RECOVERED	R	Y	Y
11-15-2024 13:59:16	2.8	MALE	60	80	-1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	9	19	60	Acinetobacter iwofii	Acinetobacter sepsis with ARDS	Tigecycline, amikacin	MECHANIAL VENTILATION	0.25	SEVERE DEFICIENCY	DEATH	R	Y	N
11-15-2024 14:00:57		MALE	70	86	-1	-2	PRESENT	PRESENT	SEVERE PNEUMONIA	10	15	34	Nogc	Viral pneumonia	Oseltamivir, amoxiclav	CPAP/ HFNC	0.29	SEVERE DEFICIENCY	RECOVERED	U	N	Y
11-15-2024 14:03:11	4	MALE	30	96	+1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	14	17	35	Nogc	Left upper lobe pneumonia with left pleural effusion	Meropenem, vancomycin	CPAP/ HFNC	0.56	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	Y
11-15-2024 14:04:46	5	FEMALE	35	95	-1 to 0	0 to -1	PRESENT	PRESENT	SEVERE PNEUMONIA	12	15	40	Nogc	Right pleural effusion with right lower lobe pneumonia	Piptaz, amikacin	CPAP/ HFNC	0.67	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
11-15-2024 14:06:05	3	FEMALE	30	98	0 to +1	0 to +1	PRESENT	ABSENT	PNEUMONIA	15	18	25	Nogc	Bronchopneumonia	Amoxiclav	NO	0.38	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
11-15-2024 14:07:33	2	FEMALE	30	96	0 to +1	0 to +1	PRESENT	ABSENT	PNEUMONIA	13	13	30	Nogc	Bronchiolitis	Amoxiclav	NASAL PRONGS/ HOOD	0.46	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	Y
11-15-2024 14:46:32	3	FEMALE	28	96	0 to +1	0 to +1	PRESENT	PRESENT	SEVERE PNEUMONIA	12	22	46	Nogc	Bronchiolitis	Amoxiclav, oseltamivir	NASAL PRONGS/ HOOD	1.07	NORMAL	RECOVERED	U	Y	Y
11-15-2024 14:48:15	4.6	FEMALE	30	98	+1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	15	13	68	Nogc	Bronchopneumonia	Piptaz, amikacin	CPAP/ HFNC	0.98	NORMAL	RECOVERED	R	N	Y
11-15-2024 14:49:20	2.8	FEMALE	30	97	+1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	9	13	40	Nogc	Viral pneumonia	Clarithromycin, amoxiclav	CPAP/ HFNC	0.87	NORMAL	RECOVERED	U	Y	Y
11-15-2024 14:52:36	2.4	FEMALE	30	96	-1	-2	PRESENT	PRESENT	SEVERE PNEUMONIA	12	17	70	MRSA	Right pleural effusion with right lower lobe pneumonia	Meropenem, vancomycin	MECHANIAL VENTILATION	1	NORMAL	DEATH	U	N	Y
11-15-2024 14:56:13	4	FEMALE	25	98	0	0	PRESENT	ABSENT	PNEUMONIA	13	10	20	Nogc	Bronchopneumonia	Amoxiclav	NO	0.35	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	Y
11-15-2024 14:57:07	2	FEMALE	30	96	-2	-2	PRESENT	ABSENT	PNEUMONIA	10	9	22	Nogc	Bronchiolitis	Amoxiclav	NO	0.88	NORMAL	RECOVERED	U	Y	Y
11-15-2024 15:10:13	0.9	MALE	50	96	-1	-1	PRESENT	ABSENT	SEVERE PNEUMONIA	12	13	50	Candida albicans	Candida sepsis with ARDS	Fluconazole, piptaz	MECHANIAL VENTILATION	0.8	NORMAL	DEATH	R	Y	N
11-15-2024 15:11:10	0.9	MALE	40	97	+1 to +2	+1 to +2	PRESENT	ABSENT	PNEUMONIA	13	15	22	Nogc	Bronchiolitis	Amoxiclav	NASAL PRONGS/ HOOD	0.7	SUBCLINICAL DEFICIENCY	RECOVERED	R	N	Y
11-15-2024 15:12:18	4	FEMALE	40	98	+1 to +2	+1 to +2	PRESENT	ABSENT	PNEUMONIA	12	10	30	Nogc	Bronchiolitis	Amoxiclav	NO	0.7	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	Y
11-15-2024 15:22:56	1.7	FEMALE	40	96	+1 to +2.	+1 to +2	PRESENT	ABSENT	PNEUMONIA	13	9	20	Nogc	Bronchopneumonia	Amoxiclav	NO	0.35	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N
11-15-2024 16:40:12	5	MALE	35	98	+1 to +2	+1 to +2	PRESENT	ABSENT	PNEUMONIA	9	8	20	Nogc	Bronchopneumonia	Amoxiclav	NO	0.4	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	N
11-15-2024 16:41:30	4	FEMALE	35	96	+2	+2	PRESENT	ABSENT	PNEUMONIA	13	9	26	Nogc	Bronchopneumonia	Amoxiclav	NO	0.5	SUBCLINICAL DEFICIENCY	RECOVERED	R	Y	N
11-15-2024 16:43:26	2.5	FEMALE	40	88	0	0	PRESENT	PRESENT	SEVERE PNEUMONIA	13	10	40	Nogc	Pneumonia with synpneumonic effusion	Meropenem	NASAL PRONGS/ HOOD	0.6	SUBCLINICAL DEFICIENCY	RECOVERED	U	N	N
11-15-2024 16:46:09	1.9	FEMALE	40	85	+1	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	10	15	40	MRSA	Left lobar pneumonia with pleural effusion	Meropenem, vancomycin	MECHANIAL VENTILATION	0.5	SUBCLINICAL DEFICIENCY	DEATH	R	Y	N
11-15-2024 16:48:32	4.6	FEMALE	36	90	-2	-1	PRESENT	PRESENT	SEVERE PNEUMONIA	8	15	54	Nogc	Right upper lobe pneumonia with synpneumonic effusion	Meropenem, amikacin	CPAP/ HFNC	1.2	NORMAL	RECOVERED	U	N	Y
11-15-2024 16:49:59	1	FEMALE	30	94	+1	+1	PRESENT	ABSENT	PNEUMONIA	14	9	25	Nogc	Bronchopneumonia with UTI	Amoxiclav	NO	1.3	NORMAL	RECOVERED	R	Y	N
11-15-2024 16:51:34	4.7	MALE	40	84	-1	+1	PRESENT	ABSENT	PNEUMONIA	9	10	25	Nogc	Bronchiolitis	Amoxiclav	NO	1.2	NORMAL	RECOVERED	U	N	N
11-15-2024 16:52:47	5	FEMALE	35	98	+2	+1	PRESENT	ABSENT	PNEUMONIA	12	8	30	Nogc	Bronchopneumonia	Amoxiclav	NO	0.6	SUBCLINICAL DEFICIENCY	RECOVERED	U	Y	Y
11-15-2024 16:54:15	0.5	FEMALE	70	88	+2	+1	PRESENT	PRESENT	SEVERE PNEUMONIA	14	15	40	Nogc	Bronchiolitis	Clarithromycin	CPAP/ HFNC	1.3	NORMAL	RECOVERED	R	Y	N
11-15-2024 16:57:20	2.9	FEMALE	50	90	0 to +1	0 to +1	PRESENT	PRESENT	SEVERE PNEUMONIA	12	12	38	Klebsiella pneumonia	Klebsiella sepsis, bronchopneumonia with synpneumonic effusion	Meropenem, amikacin	CPAP/ HFNC	1	NORMAL	RECOVERED	R	Y	N
11-15-2024 17:02:54	4.9	FEMALE	30	97	+1 to +2	+1 to +2	PRESENT	ABSENT	PNEUMONIA	13	9	30	Nogc	Bronchopneumonia	Amoxiclav	NO	0.4	SEVERE DEFICIENCY	RECOVERED	R	Y	N