"AEROALLERGEN SENSITIZATION PROFILE IN CHILDREN WITH ALLERGIC RESPIRATORY DISEASES BY EAST (ENZYME ALLERGO SORBENT TEST) METHOD IN THE AGE 5-18 YEARS: A 1 YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE CENTRE."

BY

REG NO: BM0122020

Dissertation

Submitted to the KLE Academy of Higher Education and Research, Belagavi, Karnataka

In Partial Fulfilment

of the Requirements for the Degree of

M.D. (Doctor of Medicine)

In

PAEDIATRICS

DEPARTMENT OF PAEDIATRICS

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELAGAVI, KARNATAKA

SEPTEMBER /OCTOBER 2025

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,

BELAGAVI, KARNATAKA.

Endorsement

This is to certify that the dissertation entitled **"AEROALLERGEN SENSITIZATION PROFILE IN CHILDREN WITH ALLERGIC RESPIRATORY DISEASES BY EAST (ENZYME ALLERGO SORBENT TEST) METHOD IN THE AGE 5-18 YEARS: A 1 YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE CENTRE"** is a bonafide research work done by **REG NO: BM0122020.**

Dr. DNYANESH D K M.D., L.L.B
Professor & Head
Department of Paediatrics
J N Medical College
KAHER
Belagavi, Karnataka

Dr. Dnyanesh D.K
PROFESSOR & HEAD
DEPARTMENT OF PEDIATRICS
J. N. Medical College, Belagavi

Date: 27/3/2025
Place: JNMC, Belagavi

Dr. N S MAHANTASHETTI M.D.
Principal
J N Medical College
KAHER
Belagavi, Karnataka

PRINCIPAL
Jawaharlal Nehru Medical College
Belagavi

Date: 27/3/2025
Place: JNMC, Belagavi

UNDERTAKING

I, **REG NO: BM0122020**, hereby declare that the information and data mentioned in my dissertation entitled **"AEROALLERGEN SENSITIZATION PROFILE IN CHILDREN WITH ALLERGIC RESPIRATORY DISEASES BY EAST (ENZYME ALLERGO SORBENT TEST) METHOD IN THE AGE 5-18 YEARS: A 1 YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE CENTRE"** belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date: 28-03-2025

P. Sumanth

Place: JNMC, Belagavi **(REG NO: BM0122020)**

PLAGIRISM CLERANCE

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MoE (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu rincipal@jnmc.edu

Ref No: MDC/PG/ Date: 18-03-2025

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: **"AEROALLERGEN SENSITIZATION PROFILE IN CHILDREN WITH ALLERGIC RESPIRATORY DISEASES BY EAST (ENZYME ALLERGOSORBENT TEST) METHOD IN THE AGE 5-18 YEARS - A 1 YEAR CROSS-SECTIONAL STUDY AT A TERTIARY CARE CENTRE"** has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **06%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER J.N. MEDICAL COLLEGE BELAGAVI

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BM0122020
Postgraduate Student,
2022-23 Batch,
Department of Paediatrics
J. N. Medical College, Belagavi.

ETHICAL CLERANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/94 **Date: 26/03/2023**

To,

REG NO: BM0122020
PG Student in Paediatrics
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"AEROALLERGEN SENSITIZATION PROFILE IN CHILDREN WITH ALLERGIC RESPIRATORY DISEASES BY EAST (ENZYME ALLERGO SORBENT TEST) METHOD IN THE AGE 5-18 YEARS: A 1 YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE CENTRE"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagav

ABBREVIATIONS

Abbreviations	Expansion
EAST	Enzyme Allergo Sorbent Test
BA	Bronchial asthma
AR	Allergic rhinitis
UAD	United airway disease
ISAAC	International study of Asthma and Allergies in Children
ELISA	Enzyme-linked immunosorbent assay
SPT	Skin prick test
WHO	World Health Organization
IUIS	International Union of Immunological Societies
ABPA	Allergic bronchopulmonary aspergillosis
NO_2	Nitrogen dioxide
PM	Particulate matter
APC	Antigen presenting cells
ARIA	Allergic Rhinitis and its Impact on Asthma
GRADE	Grading of Recommendations, Assessment, Development and Evaluation
MHC	Major histocompatibility complex
IgE	Immunoglobulin E
RAST	Radioallergosorbent test

MAST	Multiple allergen simultaneous test
LTRA	Leukotriene Receptor Antagonists
LABA	long-acting β-agonists
OCS	Oral corticosteroid
ICS	Inhalational corticosteroid
SLIT	Sublingual immunotherapy
Th2	T helper cells
NSAID	Non steroidal anti-inflammatory drug
GINA	Global Initiative for Asthma
SABA	Short-acting β-agonists
FENO	Fractional exhaled nitric oxide
BHR	Bronchial hyperresponsiveness
MRI	Magnetic resonance imaging
CT	Computer tomography
PFT	Pulmonary function test
NAPT	Nasal allergen provocation test
OSAS	Obstructive sleep apnoea syndrome

ABSTRACT

Introduction

Allergic conditions impact around one third of paediatric population with their prevalence rising in recent years. The tendency to develop allergies to food and airborne substances is mainly influenced by genetics. Children without family history of allergies are at 12% chances of obtaining allergies, 30% to 50% for those with one allergic parent, and even higher for those with both parents having allergies. In children and adults, the global incidence of food allergies was calculated as 4% and 1% respectively. This number is risen in the last twenty years. The study aimed to assess the sensitization profile of children in the age group 5-18 years with allergic respiratory diseases at a tertiary care centre and to identify the most common aeroallergens resulting in severe disease outcome.

Methods and Materials

Children with allergic respiratory airway diseases at a tertiary care centre, KAHER'S Dr. Prabhakar Kore hospital, Belgaum-590010, Karnataka were included in the study. The study was conducted as a cross-sectional study. The study duration was one year.

Results

The mean age of the study participants was 8.71 ± 2.89 years with majority being boys. Cold was the common clinical symptom among the study participants (n=43, 68.3%). Among the study participants in the present study, 12.7% (n=8) had one allergen and 28.6% (n=18) had two or more allergen. *Dermatophagoides*

pteronyssinus was the most common allergen (n=17, 27%). The prevalence of Allergic rhinitis was 61.9%, bronchial asthma was 30.1% and united airway disease was 8% in the present study. The most common aero allergens resulting in severe disease outcome in Allergic rhinitis, Bronchial asthma and United Airway disease were collectively found to be *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae* in the study population.

Conclusion

In conclusion, the study on aeroallergen sensitization profiles in children between age 5 to 18 years with allergic respiratory disease, assessed through EAST, provides valuable insights into the common sensitizations within this demographic area. The findings highlight the prevalence of house dust mites that contributes to respiratory symptoms, underscoring the importance of identifying sensitization in guiding effective management strategies and treatment plan for affected children. Also the most common aeroallergens resulting in severe disease outcome in the study population was identified to be house dust mite, further helping to narrow down on the causative agents to guide further plan of action.

CONTENTS

LIST OF TABLES

LIST OF FIGURE

INTRODUCTION

Allergic conditions impact around one third of paediatric population with their prevalence rising in recent years. The tendency to develop allergies to food and airborne substances is mainly influenced by genetics. Children without family history of allergies are at 12% chances of obtaining allergies, 30% to 50% for those with one allergic parent, and even higher for those with both parents having allergies.[(1)]

Allergies can be through food, skin, insect sting and respiratory allergies. Food allergy is more prevalent in individuals from industrialized nations, it occurs frequently among children when compared to adults. Severe food allergic reactions are caused due to limited number of food items like nuts including tree nuts, peanuts, milk, soy, wheat, shellfish, fish, eggs and seeds.[(2)] In children and adults, the global incidence of food allergies was calculated as 4% and 1% respectively. This number has risen in the last twenty years. [(3)] Respiratory allergies include conditions such as allergic rhinitis (hay fever) and asthma. Dust mites, pollen, spores and pet dander are some common environmental allergens which are often triggering factors for these allergies and usually present as wheezing, itching, sneezing, etc.,[(4)] Skin allergies in children often manifest as conditions like eczema (atopic dermatitis), contact dermatitis, and urticarial reactions. Eczema usually presents by red, itchy, and inflamed skin and is commonly linked with other allergic conditions like bronchial asthma (BA) and allergic rhinitis (AR). When the skin directly contacts with the allergen, it leads to contact dermatitis which shows as localized inflammation. [(5)]

The rate of allergic rhinitis has seen a continuous rise in India over the last twenty years. Important airborne allergens related to bronchial asthma and allergic rhinitis in the Indian context are house dust mites, cockroaches, pollen, and mold

spores. Study from eastern part of India discovered majority (96%) of individuals with naso-bronchial allergies were sensitive to house dust mites, particularly *Dermatophagoides pteronyssinus, Dermatophagoides farinae*, and Blomia tropicalis, with sensitivity rates of 75.06%, 63.72%, and 72% respectively. [(6)] Research from other parts of India showed a slightly lower sensitivity rates with *Dermatophagoides pteronyssinus* the predominant allergen. [(7)]

The global prevalence of bronchial asthma is rising based on the reports from The International Study of Asthma and Allergies in Children (ISAAC).[(8)] Bronchial asthma is associated with the loss of school days leading to reduced social interaction and hindering the academic success.[(9)] According to a study conducted in Northern India, the most common aeroallergen in individuals with asthma were housefly antigen and rice grain dust.[(10)] United airway disease is termed as an association for allergic rhinitis and bronchial asthma, affecting both the upper and lower respiratory tracts. [(11)]

In individuals with bronchial asthma, 80% of them have AR and in individuals with allergic rhinitis, 40% of them have BA. Hence, the terminology united airway disease. Dust mites and Parthenium leaves are the prevalent allergens in the children. [(12)]

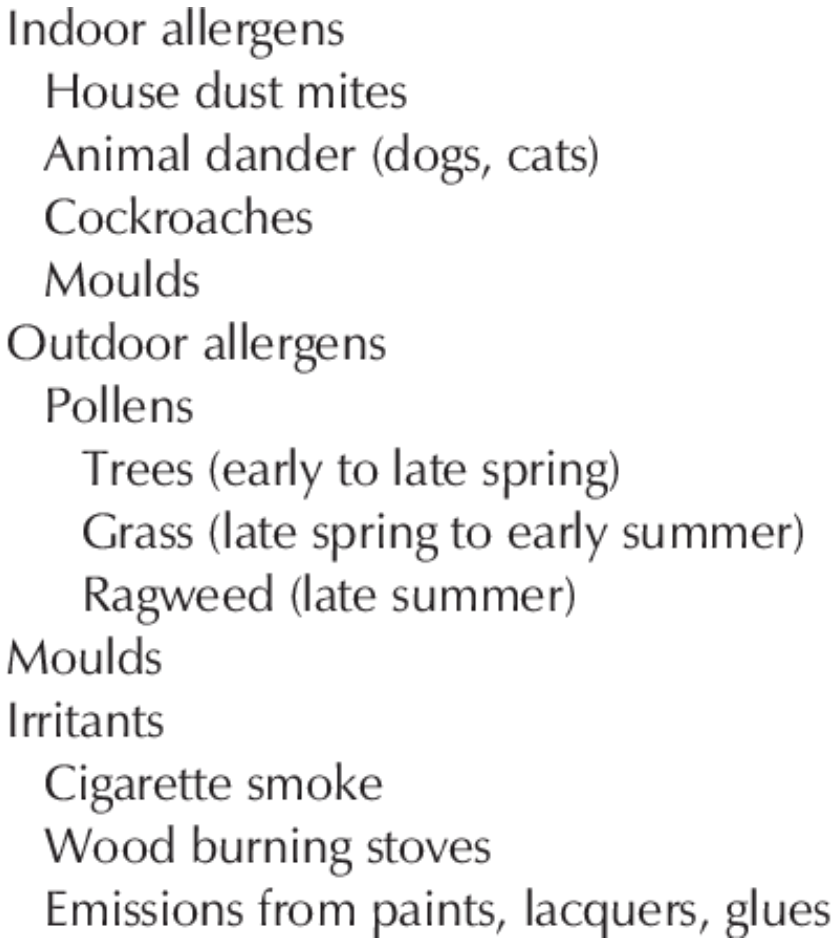

Figure 1: Common allergens and irritants

Insect sting allergies result from the body's immune response to insect's venom like bees, wasps, hornets, and fire ants. For some children, this can result in severe reactions including anaphylaxis or minor symptoms like swelling, redness, pain and in severe cases, difficulty in breathing.[13] In this study, the common respiratory allergies are dealt with in a detailed manner so as to provide clues to the most prevalent aero allergens occurring in the environment that might be responsible for allergic airway diseases. AR and BA are two diseases which can co-exist and sometimes the former precedes the latter resulting in severe disease outcome. There are various types of inhalant allergens that are commonly seen in the environment, responsible for allergic airway diseases. These include

i. Dust mite allergens - these generally thrive on human skin flakes and flourish in environments with humidity levels above 50%. To mitigate dust mite exposure, it is essential to remove dust mite habitats and reduce indoor humidity. The bedroom is a critical area for dust mite allergen exposure and should be the primary focus for control measures. For individuals with dust mite-sensitive asthma, environmental control is the most effective treatment. Implementing these allergen avoidance strategies significantly reduces the severity of asthma symptoms and decreases the reliance on symptomatic medications. [(14)]

ii. Cockroach allergen - Exposure to cockroach allergens is mainly seen in inner city dwellings and buildings hence clean housekeeping practices and use of insecticides can help.

iii. Animal dander -Ideally, for patients sensitized to animals, pet-free homes are advised. For example, the cat allergens are secreted from the sebaceous glands present in skin, with a size of 2 – 4 μm, and it gets suspended in the air for a longer time. Dander may remain in the carpets and furniture for extended period even after removal of the cat. To reduce the dander, the pets to be bathed every day.[(15)]

iv. Mold –Penicillium and aspergillus are the common indoor molds and Cladosporium & Alternaria are the common outdoor molds which may also be present indoors. All mold types thrive in humid conditions, with prime growth areas being bathrooms, kitchen, and basements with bad ventilation. Forced air heating systems with central humidifiers may become significant mold reservoirs without proper maintenance.[(16)] For individuals confirmed to be sensitive to mold, it is crucial to maintain low humidity levels, ideally around 35%, and certainly below 50%. Measures to control mold include enhancing

ventilation completely in the home, particularly in the bathrooms and basements, venting dryers to exterior; eliminating carpets in basements or previously flooded areas; and inspecting walls for moisture infiltration.

v. Pollen-In the spring period, tree pollens are the common pollens and from late spring to early summer, grass pollens and in the late summer, ragweed pollens are commonly present. During the early morning, the pollen levels are increased. Windows should be closed in this duration to reduce the entry of pollens to home.

vi. Irritants-It has long been established that exposure to irritants like cigarette smoke significantly affects severity and incidence of respiratory allergies. Parents of children with respiratory allergies should refrain from smoking indoors. Additionally, children who live in homes with wood-burning stoves tend to experience more frequent respiratory infections. [17]

Figure 2: Types of inhalant allergens

Enzyme allergo sorbent test is an immunological assay utilized in quantification of specific IgE antibodies in serum, specifically in relation with allergens such as food and inhalant substances. This test is particularly valuable in the diagnosis of allergic conditions, allowing for the identification of sensitization to a range of allergens. EAST operates on principle of enzyme-linked immunosorbent assay (ELISA), where allergen extracts are immobilized on test strips.[18] The skin prick test (SPT) is commonly used method in diagnosing allergies due to its accuracy, ease and cost-effectiveness, which involves the pricking of the skin, usually forearm, using lancet containing an allergen extract, allowing multiple allergens to be tested in 15 to 20 minutes.[19] SPT is invasive and EAST is non-invasive which is an important factor when testing the children. EAST test is more reliable as it uses IgE for detection, while SPT results can interfere in children who were on antihistamines. Although, SPT is widely regarded as sensitive and cost-effective diagnostic tool for allergen sensitization, these limitations were considered and hence the present study used EAST for diagnosis.

AIMS AND OBJECTIVES

Primary objective

- To assess the sensitization profile of children in the age group 5-18 years with allergic respiratory diseases at a tertiary care centre.

Secondary objective

- To identify the most prevalent aero-allergens that might result in severe disease outcome.

REVIEW OF LITERATURE

Burden of the disease

Allergic airway disease encompasses various respiratory ailments marked by inflammation and heightened sensitivity of the air passages provoked by exposure to allergens. Predominant among these conditions are AR, also called as hay fever, and BA. The prevalence of allergic airway diseases has notably surged in the recent past due to environmental influences like tobacco smoke, air contaminants, and indoor & outdoor allergens.

A study estimated the disease burden as 262 million individuals affected with asthma, with the illness claiming the lives of around half a million people annually. [20] While asthma can impact individuals of all ages, adolescents may experience the most significant challenges associated with the condition.[21] The phenomenon of allergic airway diseases is common among the paediatric age group with allergic rhinitis occurring at rates varying from 0.8% to 14.9% among 6 to 7 years and 1.4 to 39.7% among 13 to 14 years. According to recent most studies, AR prevalence increased from 8.5% to 14.6% in kids living in under-developed and developing countries. Symptoms typically seen to manifest within 20 years of age, with almost half (40%) before 6 years of age.[22] A study conducted on incidence of BA in children in India revealed significant variations across states. Maharashtra reported the highest prevalence at 24.8%, followed closely by Karnataka at 21.5% and Delhi at 21.3%. Other states, including Andhra Pradesh (15.5%), West Bengal (15.3%), and Tamil Nadu (14.9%), also exhibited notable rates. In contrast, Gujarat, Madhya Pradesh, and Rajasthan showed lower prevalence rates ranging from 11% to 12%.

These findings highlight the need for targeted asthma management and awareness programs tailored to specific regional challenges in India. [23]

AR and BA pose significant challenges to public health and economic well-being globally due to their widespread occurrence, associated health issues, and reduces the quality of life.[24] Aeroallergens constituting pollen & fungal spores significantly contribute to allergic respiratory diseases in children, with distinct allergen profiles observed across different geographical regions. While Western populations commonly encounter allergies to grass, rye, and pollen, children in the Indian subcontinent frequently exhibit sensitization to house dust mites, cockroaches, and animal dander.[25] Notably, infants and young children are often the initial recipients of sensitization present continuously in environment, resulting in symptomatology.[26]

Brief evolution

Although sporadic reports with elevated incidence of BA surfaced during 1960s, it wasn't until 1969, with Smith et al.'s study in Birmingham, UK, that convincing evidence emerged. They noted a significant uptick in asthma cases among school children, many of whom tested positive for dust mite allergies.[27] Despite the widespread acknowledgement today of asthma's rise in Western countries, this trend only became evident in the 1990s. By 1995, it was recognized that asthma prevalence and hospitalizations surged, particularly among children exposed to various allergens prevalent in their climates or living conditions. The surge in hay fever (allergic rhinitis) cases around the late 19th century was likely fueled by a convergence of factors including improved hygiene practices and agricultural changes leading to heightened pollen levels. While asthma in children began to rise notably around the

1960s, reaching epidemic proportions by 1990, the shift towards indoor lifestyles played a pivotal role. Variables like increased exposure to indoor allergens, dietary changes, and reduced physical activity likely contributed.[28]

Common Aeroallergens

Indoor allergens

i. Mites

House dust mites are a major cause of allergic diseases worldwide, playing a key role in triggering conditions like allergic rhinitis, eczema, and asthma. According to the World Health Organization and the International Union of Immunological Societies' Allergen Nomenclature Database, mite allergens are classified into 33 distinct groups. A large portion of these allergens originates from mite feces, with Der p 1 being the first allergen identified, typically found at concentrations around 10 mg/ml. These fecal particles, which measure between 10 to 40 micrometers, can become airborne when disturbed, leading to exposure primarily through inhalation. However, oral intake of mite-contaminated foods—particularly those made with infested wheat flour—has also been associated with allergic reactions, including anaphylaxis, a phenomenon sometimes described as "pancake syndrome." The most common dust mite species include Dermatophagoides pteronyssinus and Dermatophagoides farinae, along with others like Dermatophagoides microceras, Euroglyphus maynei, Blomia tropicalis, and several storage mites such as Glycyphagus domesticus, Lepidoglyphus destructor, Acarus siro, and Tyrophagus putrescentiae.

ii. Cockroach

The association between cockroach allergens and allergic reactions was first documented in 1964 through positive skin test results. Research has since established a strong connection between cockroach allergy and respiratory conditions such as allergic rhinitis and asthma. The two main species implicated in these allergic responses are the German cockroach (Blattella germanica) and the American cockroach (Periplaneta americana). As per the World Health Organization/ International Union of Immunological Societies (WHO/IUIS) Allergen Nomenclature Database, 12 distinct allergen groups have been identified from cockroach sources.

iii. Mammalian allergen

Allergens from mammals are mainly synthesized in the liver and various secretory glands. These allergens are present on the skin and in biological fluids such as saliva, urine, and blood. Most major animal allergens belong to the lipocalin protein family, except for the prominent cat allergen Fel d 1. These proteins tend to adhere to fur and are easily dispersed into the environment, accumulating on various surfaces such as fabrics, carpets, and mattresses. This widespread distribution underscores the significance of animal protein allergies as a public health concern.

iv. Cat Allergens

Fel d 1 is a 38 kDa tetrameric glycoprotein, structurally related to uteroglobin, and is responsible for triggering IgE-mediated allergic responses in more than 90% of individuals allergic to cats. It is secreted by the sebaceous, salivary, and anal glands and then spread onto the cat's fur through grooming. Although most airborne Fel d 1 is bound to larger particles, a fraction is found in smaller particles that can remain

suspended in the air for several days, prolonging exposure. In school settings, the concentration of cat allergens is often linked to how many students live with cats. Other recognized cat allergens include Fel d 2, Fel d 4, and Fel d 7.

v. Dog Allergens

Among the six identified dog allergens, four—Can f 1, Can f 2, Can f 4, and Can f 6—are lipocalins. Approximately three-fourth of individuals allergic to dogs have IgE antibodies specific to Can f 1, which can be found in homes with and without dogs. Variability in Can f 1 levels exists among different dog breeds, but no hypoallergenic breeds have been proven. Can f 5 is also significant, affecting a considerable number of dog-allergic patients.

vi. Rodent Allergens

Allergies to mice and rats present a notable occupational health risk, particularly among laboratory workers and animal caregivers. Rodent exposure in domestic settings is also common, particularly in urban children with asthma. Key rodent allergens, including Mus m 1 from mice and Rat n 1 from rats, are mainly present in their urine and are typically attached to fine particles that can stay suspended in the air. Children whose parents work in environments with rodent exposure tend to have higher sensitization rates. Gaining insight into these allergens plays an important role in effectively managing allergy-related conditions.

vii. Fungi

More than 15 types of fungi are commonly found in urban households, with Cladosporium, Penicillium, Aspergillus, and Alternaria being the most prevalent. Cladosporium and Alternaria, in particular, are well-known sources of outdoor

allergens, and their exposure has been associated with the onset of asthma, allergic rhinitis, and severe asthma flare-ups, some of which can be life-threatening.

Alternaria alternata shows a sensitization rate of approximately 5% and is strongly linked to respiratory allergic conditions. Its primary allergen, Alt a 1, is present in over 90% of sensitized individuals. Other notable allergens from this fungus include Alt a 2 (a 25 kD aldehyde dehydrogenase) and Alt a 5 (an enolase), which are recognized by 20% to 50% of those affected.

Cladosporium herbarum is another major airborne mold allergen, commonly detected in both indoor and outdoor environments. While Alternaria thrives in humid climates, Cladosporium is more dominant in cooler regions. Its major allergen, Cla h 8 (a NADP-dependent mannitol dehydrogenase), is detected by IgE in 57% of Cladosporium-allergic individuals. Another allergen, Cla h 6 (an enolase), is identified by about half of sensitized patients.

Aspergillus fumigatus is a heat-tolerant fungus found worldwide and is the leading cause of allergic bronchopulmonary aspergillosis (ABPA). It also contributes to asthma in individuals with hypersensitivity, marked by the presence of specific IgE antibodies against A. fumigatus. The main allergen, Asp f 1, an 18 kD ribotoxin, is recognized in 85% of sensitized patients, while Asp f 2 and Asp f 3 also show significant reactivity.

Penicillium species, particularly P. citrinum and P. chrysogenum, are common indoor molds linked to allergic responses. Pen ch 13 and Pen ch 18, two major allergens from P. chrysogenum, elicit specific IgE responses in 88% and 82% of sensitized individuals, respectively. In contrast, allergens from P. citrinum show

generally lower reactivity, with Pen c 3 (an 18 kD membrane protein) being the most reactive .[29]

viii. Tobacco Smoke

Elevated serum cotinine levels show a significant association with IgE sensitization to various allergens, includes cockroaches, grass pollen, and some foods. These associations appear to be dose-dependent, though they vary among different allergens specifically in children.

ix. Cooking Gas and Nitrogen Dioxide

Short-term exposure to nitrogen dioxide (NO_2), particularly from gas stoves, has been moderately linked to respiratory problems and reduced lung function in children, though these effects are not consistently observed in women. In contrast, prolonged exposure to NO_2 has shown a stronger association with respiratory symptoms in children, including decreased lung capacity and increased instances of chronic cough, bronchitis, and eye irritation, whereas such patterns are less evident in adults.

x. Fragrance and Preservatives

Cosmetic products frequently contain preservatives and fragrances that are common triggers for allergic contact dermatitis. Although certain terpenes used for fragrance are not naturally allergenic, they can form allergenic compounds upon exposure to air. To address this, the European Union requires labeling of 26 specific fragrance ingredients known to cause allergic responses.

xi. Chemical Allergens

Children are particularly vulnerable to respiratory and allergic conditions linked to exposure to specific indoor chemicals. These include formaldehyde released from composite wood products, plasticizers from flexible plastics, and emissions from fresh paint. Even at low levels, indoor pollutants like propylene glycol and glycol ethers have been associated with increased occurrences of asthma, rhinitis, eczema, and IgE sensitization in young children. Furthermore, elevated humidity in buildings with concrete floors and the release of 2-ethyl-1-hexanol—a byproduct of plasticizer breakdown in damp conditions—can worsen asthma symptoms.

Outdoor allergens

i. Pollens

Pollen is a major trigger for allergies, impacting nearly 40% of people with allergic conditions. Due to their lightweight and water-soluble nature, pollen grains are easily dispersed in the air and are produced in large volumes. Common sources include grasses, trees, and weeds, with ornamental plants increasingly contributing as well. Most pollen particles, measuring between 20 to 60 micrometers, tend to affect the upper airways, causing typical allergic symptoms. However, much smaller particles—less than 3 micrometers—can penetrate deeper into the lungs, potentially causing lower respiratory tract issues.

ii. Grass

Grass pollen contains 20 to 40 distinct antigens, which are grouped into eight categories based on their immune responses. The most important among these are Group I allergens, which are about 3 micrometers in size and originate from the outer

layers of the pollen grain. These allergens trigger reactions in approximately 90% to 95% of individuals with grass pollen allergies during skin tests. Groups II and III are also reactive in 60% to 70% of these cases. Additionally, grass pollen has been associated with contributing to atopic dermatitis, particularly in children.

iii. Tree Pollen

Tree pollen allergies are mainly caused by species within the Fagales and Pinales orders. Among them, birch pollen contains the major allergen Bet v1, which was the first tree pollen allergen gene to be cloned. Since then, a total of 53 allergens from tree pollen have been identified. Due to extensive cross-reactivity among different tree pollens, birch pollen testing is commonly used to diagnose related allergies. Bet v1 also shares similarities with certain proteins in apples, leading to oral allergy symptoms. In Japan, Japanese cedar is a key allergenic tree, while in Mediterranean regions, olive tree pollen is a major contributor to allergic reactions.

iv. Air pollutants

Air pollution, especially from particulate matter (PM), comprises a combination of airborne solid and liquid particles along with various harmful gases and chemicals. These pollutants are largely produced through the burning of fossil fuels such as coal and oil during construction work, farming operations, energy production, and household activities like cooking and heating. Notably, diesel engine emissions are responsible for over 80% of these fine particles.

The physical characteristics and chemical makeup of PM—such as size and shape—determine how deeply it can enter the respiratory system and the extent of its health impact. These particles can travel long distances before being cleared from the

atmosphere by mechanisms like rainfall, gravity, or merging with other particles. According to the World Health Organization's 2005 air quality guidelines, daily exposure to PM2.5 should not exceed 25 µg/m³, and PM10 should remain below 50 µg/m³. Despite this, many urban areas regularly surpass these thresholds, and no universally safe exposure level has been established.

Global research has identified strong links between PM exposure and an increased risk of asthma, allergic rhinitis, and heightened sensitivity to pollen, although no similar connection has been observed with atopic dermatitis. People living near major roads—typically within a 100-meter radius—are more prone to respiratory allergies, diminished lung function, and greater sensitization to environmental allergens. Additionally, diesel particles may enhance allergic responses by acting as mucosal adjuvants, facilitating the immune system's reaction to new airborne allergens. [30]

Table 1: Comparison of western and Indian aeroallergens

Variables	Western	India
Tree pollen	Oak, Maple, Pine, Birch, Hickory, Cottonwood, Pecan, Cedar	Acacia, Prosopis juliflora (Vilayati Babul), Azadirachta indica (Neem), Holoptelea (Indian Elm), Morus (Mulberry)
Grass pollen	Timothy grass, Bermuda grass, Kentucky bluegrass, Ryegrass, ragweed	Cynodon dactylon (Bermuda grass), Parthenium hysterophorus (Congress grass), Saccharum (Sugarcane)
Weed pollen	Ragweed, Sagebrush, Pigweed, Dockweed	Parthenium, Artemisia (Mugwort), Amaranthus, Ricinus (Castor), Chenopodium
Fungal spores-Outdoor	Alternaria, Cladosporium, Stachybotrys (Black mold), Fusarium	Alternaria, Aspergillus, Cladosporium, Penicillium
Fungal spores-Indoor	Aspergillus, Penicillium, Stachybotrys	Aspergillus, Rhizopus, Candida
Mites	Dermatophagoides pteronyssinus, Dermatophagoides farinae	Dermatophagoides pteronyssinus, Dermatophagoides farinae, Blomia tropicalis
Pollutants	Low PM2.5 and PM10	High PM2.5 and PM10
Pet allergens	Cat, Dog, Horse dander	Cat, Dog, Cow dander
Cockroach	Periplaneta americana (American cockroach), Blatella germanica (German cockroach)	Periplaneta americana (American cockroach), Blatella germanica (German cockroach)

Allergic rhinitis

Definition

"Allergic rhinitis is a symptomatic disorder of nose induced after exposure to allergens via IgE-mediated hypersensitivity reactions, which are characterized by 4 cardinal symptoms of watery rhinorrhoea, nasal obstruction, nasal itching and sneezing (Min YG)". [31]

Allergic rhinitis is believed to impact around 400 million people globally, with children making up nearly 40% of those affected. [32]

In India, approximately 7.7% of children aged 6 to 7 years and 23.5% of those aged 13 to 14 years are affected by allergic rhinitis.[33]

Allergic Rhinitis-Overview

Allergic rhinitis is a long-term condition that affects 20-30% of children. It involves inflammation of the nasal lining. Children with allergic rhinitis are three times more likely to develop asthma as they get older and this condition is most common in late childhood.

While allergic rhinitis may not be as severe as some other medical conditions in terms of sickness and death rates, its impact on individuals and society is significant due to the considerable burden it places on healthcare resources and the economy.[34] Allergic rhinitis greatly impacts the well-being of many individuals, affecting their ability to sleep well and think clearly while leading to irritability and exhaustion. Furthermore, it hinders academic and professional performance, especially during times of heightened pollen activity.[35] Over the past 50 years, the incidence of allergic rhinitis has increased globally, with some studies reporting

prevalence rates reaching up to 40%. Although genetics contribute significantly to its inheritance, environmental changes, dietary shifts, and alterations in the microbiome are considered major factors in this rise. Key risk factors include a family history of atopic conditions and serum IgE levels exceeding 100 IU/ml by the age of six. Early life exposures—or lack thereof—also influence susceptibility to allergic diseases. Children born to mothers who smoke heavily during pregnancy and continue smoking throughout the child's first year, as well as those frequently exposed to indoor allergens, face a higher risk. A critical period during infancy exists when genetically susceptible children are more prone to developing allergies. Babies born via cesarean section may be at greater risk for allergic rhinitis and atopy due to limited exposure to maternal microbiota during delivery. Additionally, toddlers with elevated antibodies against cockroach or mouse allergens are more likely to develop conditions such as wheezing, allergic rhinitis, and eczema upon future exposures. Research also suggests that experiencing three or more episodes of runny nose in the first year of life may be linked to allergic rhinitis by the age of seven

Interestingly, early exposure to allergens might protect against developing atopy, and prolonged breastfeeding has been shown to be beneficial. Introducing foods like wheat, rye, oats, barley, fish, and eggs early in a child's diet is linked to decrease in risk of respiratory allergic airway diseases. Conversely, a lower diversity of gut microbiota during infancy is linked to a higher risk of allergic diseases in school-aged children.[35]

Classification

Traditionally, AR is categorized based on the its nature and chronic distribution as:

- Seasonal (acute) and
- Perennial (chronic).

Seasonal allergic rhinitis is typically caused by outdoor allergens such as pollen, while perennial allergic rhinitis is generally linked to indoor allergens like dust mites, cockroach allergens, pet dander, or mold. However, in some areas, pollen may contribute to year-round symptoms when it becomes trapped indoors in items like carpets, furniture, or bedding, even after the pollen season ends. Additionally, perennial rhinitis symptoms may not always be present throughout the year, and individuals who are sensitized to several allergens may continue to exhibit symptoms beyond seasonal exposure. Patients with perennial allergic rhinitis can also experience seasonal flare-ups when exposed to pollen. Due to these overlapping patterns, the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines, introduced in 2001, proposed replacing the terms 'seasonal' and 'perennial' with 'intermittent' and 'persistent.' The classification of allergic rhinitis was also expanded to include disease severity based on symptom intensity as mild, moderate and severe based on its effects on daily activities, work or school performance, and sleep quality.

ARIA Classification

The Allergic Rhinitis and its Impact on Asthma (ARIA) initiative originated from a World Health Organization (WHO) workshop held in 1999, with its first set of findings released in 2001. ARIA introduced a new classification for allergic rhinitis (AR), categorizing it as mild or moderate-to-severe and as intermittent or persistent.

The initiative emphasized the close link between allergic rhinitis and asthma, as well as the importance of patient-centered care. The variety of available treatments for AR has led to inconsistencies in clinical practices and uncertainty regarding the effectiveness and limitations of various therapies across the globe. In 2010, ARIA updated its guidelines to include clinical recommendations for managing both AR and asthma, using the GRADE (Grading of Recommendations, Assessment, Development and Evaluation) framework. These guidelines have been adopted in over 50 countries. A decade after ARIA's initial WHO report, it remains essential to review its progress, address ongoing clinical and research gaps, and support the European Union's 2011 initiative that prioritizes allergy and asthma care in children.

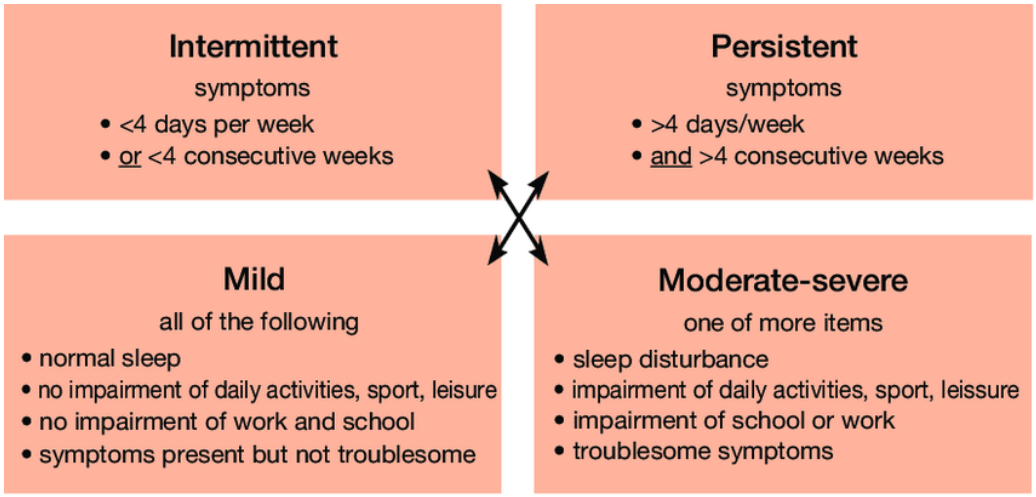

Figure 3: ARIA Classification

Pathogenesis

i. Allergen sensitization

Antigen-presenting cells (APCs), such as dendritic cells found on mucosal surfaces, process allergens and present allergen-derived peptides on major histocompatibility complex (MHC) class II molecules. This MHC-antigen complex interacts with T-cell receptors on naïve CD4+ T cells, prompting their development into allergen-specific Th2 cells. These Th2 cells, upon activation, secrete cytokines

that support B cell isotype switching for IgE production and encourage the expansion of eosinophils, mast cells, and neutrophils. The allergen-specific IgE produced then binds to high-affinity IgE receptors located on mast cells and basophils.

ii. Early and late reactions

Allergic rhinitis symptoms occur in two phases after allergen exposure. The early phase begins within 30 minutes and is marked by sneezing and nasal discharge, which usually diminish quickly. This immediate response is primarily caused by mast cells, which initiate a Type I hypersensitivity reaction by releasing chemical mediators such as histamine, prostaglandins, and leukotrienes. The late-phase reaction typically develops around six hours later and involves progressive nasal congestion. This delayed response results from eosinophil recruitment triggered by mediators from the initial phase. Inflammatory cells like eosinophils, mast cells, and T cells then accumulate in the nasal lining, causing tissue inflammation and remodeling, which leads to persistent nasal blockage—a common symptom in allergic rhinitis.

iii. Neurogenic inflammation

Neurogenic inflammation arises when toxic proteins released by eosinophils injure the respiratory epithelium, leading to the exposure of underlying nerve endings. These exposed sensory nerves can then be activated by various non-specific triggers, resulting in stimulation of both the incoming (afferent) and nearby outgoing (efferent) nerve fibres—a process referred to as the retrograde axonal reflex. This neural response leads to the release of neuropeptides like substance P and neurokinin A, which contribute to airway smooth muscle contraction, increased mucus production from goblet cells, and fluid leakage from capillaries.

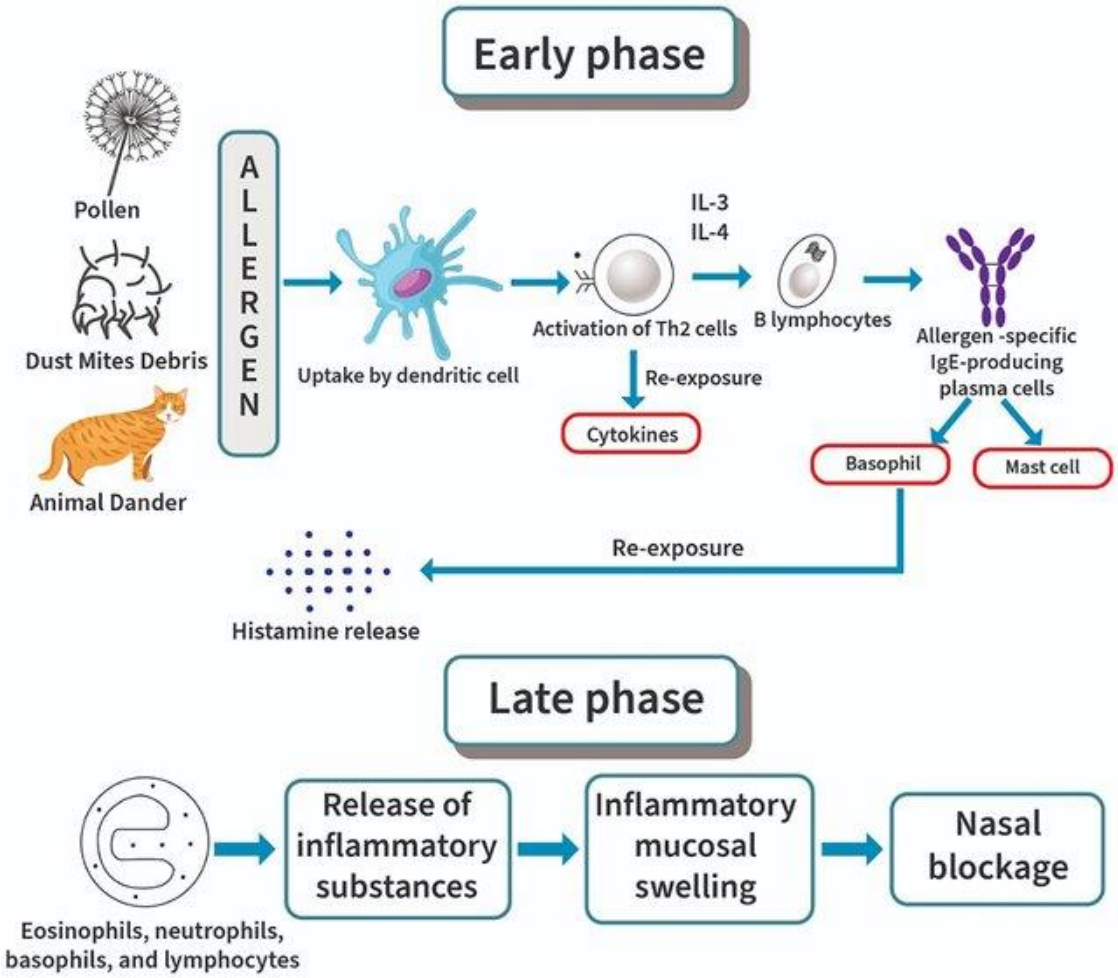

Figure 4: Pathophysiology of Allergic rhinitis

iv. Non-specific Hyper-responsiveness

Non-specific hyper-responsiveness is a hallmark of allergic inflammation. The infiltration of eosinophils and resulting injury to the nasal lining make it excessively sensitive to everyday stimuli, triggering symptoms such as nasal congestion, sneezing, itching, and runny nose. Unlike IgE-mediated immune reactions, this type of responsiveness is non-immune in nature. Individuals with allergic rhinitis not only react to particular allergens but also show increased sensitivity to irritants like cigarette smoke or cold, dry air.

Diagnosis of Allergic Rhinitis

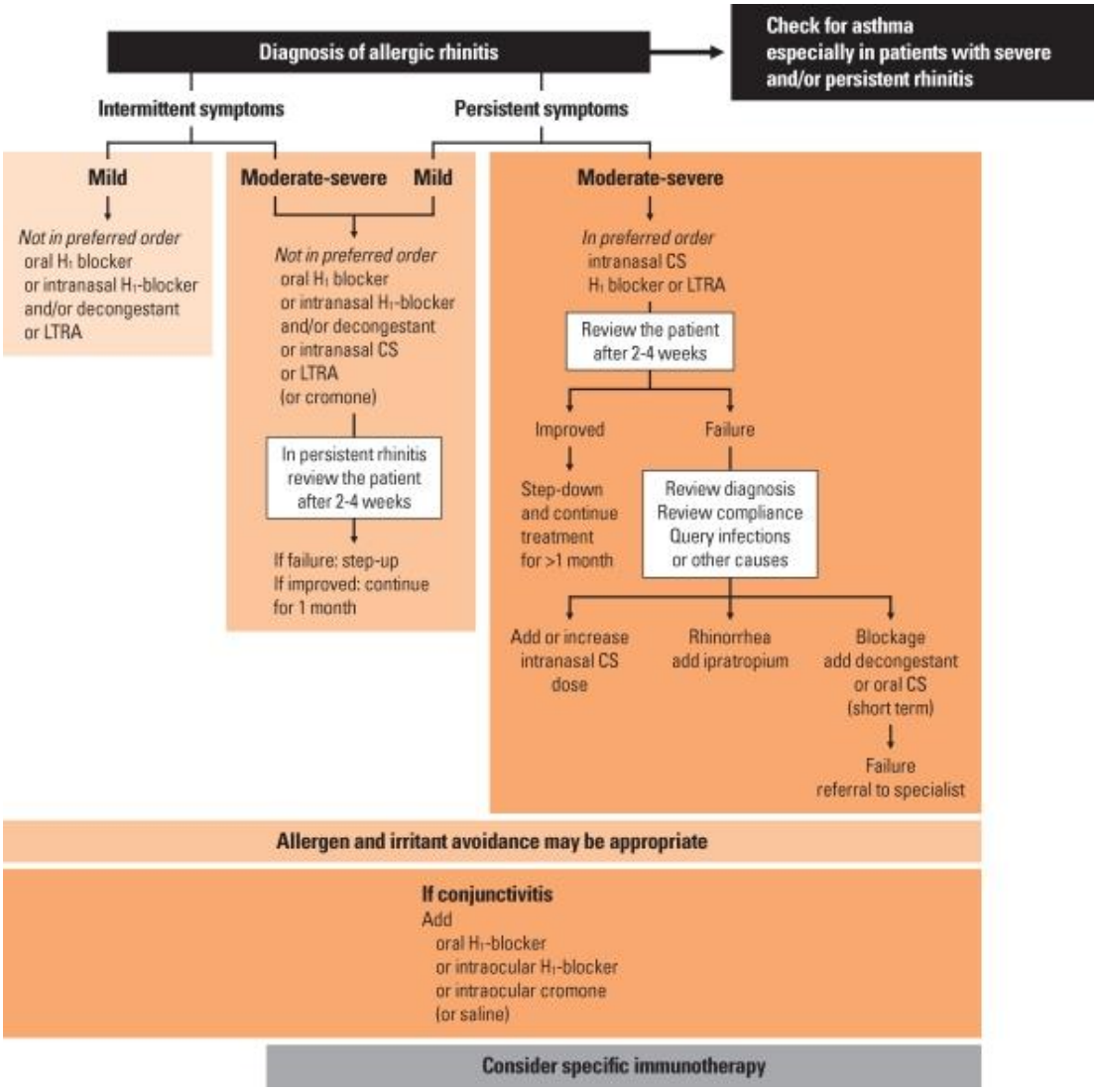

Figure 5: Diagnosis of Allergic Rhinitis

Diagnosing allergic rhinitis (AR) is based on a typical clinical history and confirmatory diagnostic tests. AR should be considered when two or more symptoms—such as watery nasal discharge, sneezing, nasal blockage, or itching—persist for at least an hour on most days. The condition's severity should be assessed following the ARIA (Allergic Rhinitis and its Impact on Asthma) guidelines, and confirmation should be obtained through skin prick testing or measurement of allergen-specific IgE in the blood. On the other hand, symptoms like one-sided nasal

blockage, thick or discolored nasal discharge, postnasal drip with mucus, facial pain, frequent nosebleeds, or loss of smell are typically not indicative of AR.

Skin Testing for AR

Skin testing plays a vital role in identifying the allergens responsible for allergic rhinitis (AR). Several techniques are used for this purpose, such as scratch testing, prick or puncture testing, intradermal testing, and patch testing, with the skin prick test being the most widely utilized in routine practice. Despite its widespread use, skin testing can yield false-positive or false-negative outcomes, meaning a positive result may not always correlate with clinical symptoms in the nasal mucosa. Interpretation of these results remains controversial, and the threshold for a positive test can differ between healthcare settings. Additionally, factors like age, test site, and the use of medications—especially antihistamines—can influence test accuracy. Skin testing may also be unsuitable for individuals with certain skin disorders. Nevertheless, it remains an important tool in diagnosing AR.

Although the radioallergosorbent test (RAST) was the first method created to detect specific IgE in serum, its use has declined due to the need for radioactive substances, expensive equipment, and its inability to measure multiple allergens simultaneously. In contrast, the multiple allergen simultaneous test (MAST) has gained popularity due to its advantages. MAST uses a photoreactive agent instead of radioactive materials, is less costly, can detect several allergens at once, and is not influenced by medications like antihistamines, making it suitable for patients with dermographism. However, MAST's sensitivity is lower than that of the skin prick test. Still, a study by Finnerty et al. demonstrated MAST had concordance rates of 66.5% and 78.5% for reaction sizes of ≥3 mm and ≥5 mm, respectively, suggesting it could

be a viable alternative. Another development is the capsulated hydrophilic carrier polymer (CAP) system, a more advanced in vitro method that, like MAST, detects specific IgE but with enhanced precision. This technique uses a solid-phase cellulose polymer that binds antigens to its inner surface, enabling more accurate quantification of allergen-specific responses.

Treatment of AR

i. Avoidance:

Completely avoiding indoor allergens like house dust mites can be difficult in everyday settings. According to the 2001 ARIA guidelines, the evidence supporting allergen avoidance methods was considered to be of low quality. This position was reinforced in the 2008 ARIA update, which highlighted the insufficient evidence for the effectiveness of strategies aimed at reducing exposure to house dust mites or pet allergens. However, in cases of occupational allergic rhinitis, strict allergen avoidance remains crucial. The most reliable and effective treatment in such cases is to eliminate exposure to the specific allergen responsible for triggering symptoms.

ii. Pharmacological Treatment

Pharmacological treatment is typically administered using a stepwise strategy based on the intensity and persistence of symptoms. The 2008 ARIA guidelines brought notable updates compared to the 2001 recommendations. These included endorsing leukotriene receptor antagonists for all types of allergic rhinitis, favoring second-generation antihistamines over first-generation ones due to their improved safety, and identifying topical corticosteroids as the most effective option for managing symptoms in both adults and children.

iii. Oral Antihistamines

First-generation antihistamines, in use since the 1940s, are associated with adverse effects like drowsiness, impaired memory, and reduced psychomotor performance, which can complicate clinical management. Second-generation antihistamines, on the other hand, are less likely to cross the blood-brain barrier, resulting in fewer central nervous system side effects. As a result, the 2008 ARIA guidelines favor second-generation antihistamines over older versions. These drugs are effective in relieving symptoms such as sneezing, nasal itching, watery discharge, and eye discomfort, though they are not as effective for nasal congestion. Earlier second-generation options like terfenadine and astemizole are now rarely used due to serious cardiac side effects, including QT interval prolongation, especially when combined with certain antibiotics or antifungals that inhibit the CYP3A4 enzyme.

iv. Intranasal Antihistamines:

Intranasal antihistamines are effective in managing symptoms like nasal itching, sneezing, and runny nose. However, they tend to be less potent than intranasal corticosteroids and offer little benefit for eye-related symptoms. In cases of seasonal allergic rhinitis where oral antihistamines are insufficient, using intranasal azelastine twice daily can help alleviate symptoms. Some individuals may, however, experience minor side effects such as drowsiness and a metallic aftertaste.

v. Intranasal Corticosteroids

Intranasal corticosteroids are known for their low risk of systemic side effects due to limited absorption into the bloodstream. These drugs work by crossing cell membranes and binding to steroid receptors in the cytoplasm. This steroid-receptor

complex then interacts with DNA in the cell nucleus, altering protein production and regulating transcription factors to produce anti-inflammatory effects. They are effective in suppressing both early and late-phase allergic responses by lowering IgE levels and reducing eosinophil activity through inhibition of cytokines like IL-4, IL-5, and IL-13. A noticeable decline in eosinophil and basophil counts is usually observed within one week of starting therapy, with symptom relief typically beginning around seven hours after administration and peaking within two weeks.

Currently used intranasal corticosteroids include budesonide, triamcinolone acetonide, fluticasone propionate, mometasone furoate, and fluticasone furoate. While their therapeutic effects are generally comparable, they differ in how much of the drug is absorbed systemically. Most are metabolized in the liver via the first-pass effect, which further reduces systemic exposure. These medications are considered safe, and short-term use of mometasone (200 µg) or budesonide (400 µg) in children aged 7–12 has not shown any significant impact on growth. However, long-term use of beclomethasone dipropionate may slow growth, whereas extended use of mometasone and fluticasone appears to be growth-neutral. Additionally, intranasal corticosteroids have been found to alleviate symptoms in patients with coexisting asthma and allergic rhinitis, with evidence showing that beclomethasone can help reduce airway hyperresponsiveness and asthma symptoms.

vi. Leukotriene Receptor Antagonists (LTRAs)

Leukotrienes play a significant role in allergic reactions, and leukotriene receptor antagonists (LTRAs) have already proven effective in treating asthma. More recently, their usefulness in managing allergic rhinitis (AR) has garnered attention, especially following the updated 2008 ARIA guidelines that re-evaluated their

therapeutic potential. This renewed interest aligns with the “one airway, one disease” concept, leading to increased research into the benefits of LTRAs for AR. Commonly used LTRAs include pranlukast (Onon®) and montelukast (Singulair®). It’s important to exercise caution with pranlukast, as it is metabolized by liver enzyme CYP3A4, which may elevate drug levels when combined with medications like terfenadine, astemizole, ketoconazole, or erythromycin.

Montelukast has demonstrated effectiveness in relieving nasal and eye symptoms in seasonal AR and has shown similar improvement in nasal blockage as loratadine. However, the benefit of using montelukast together with loratadine remains inconclusive. Combining montelukast with cetirizine six weeks before pollen season has been found to reduce the likelihood of seasonal symptom flare-ups. While the therapeutic effects of LTRAs are comparable to antihistamines, they tend to be less effective than intranasal corticosteroids for treating seasonal allergic rhinitis.

vii. Anti-IgE Antibody

Omalizumab is a recombinant humanized monoclonal antibody designed to target immunoglobulin E (IgE). By binding to free IgE in the bloodstream, it disrupts the interaction between IgE and mast cells or eosinophils, leading to a decrease in circulating IgE levels. This action also reduces inflammation in the nasal passages and bloodstream and lowers the expression of FcεRI receptors on the surface of mast cells and eosinophils. In individuals with severe seasonal allergic rhinitis (AR), a 12-week course of omalizumab at a dosage of 300 mg—administered before and during pollen season—resulted in notable symptom improvement.

Although some adverse effects, such as headaches, sinus infections, and upper respiratory tract infections, were observed, their frequency was similar to that seen in placebo groups. A few patients developed localized hives at the injection site, which usually resolved on their own or responded to antihistamines. While anti-IgE therapy like omalizumab has shown promise in treating severe asthma, its role in AR management is still debated due to concerns about potential anaphylaxis and the high cost of treatment.

viii. Immunotherapy

Immunotherapy is the only treatment that directly targets the root cause of allergies by gradually desensitizing the immune system and inducing tolerance to specific allergens. Originally created to manage seasonal allergic rhinitis (AR) triggered by pollen, its use has since broadened to treat allergies related to insect stings, dust mites, pet dander, and mold. This therapy typically involves subcutaneous injections of allergen extracts, with the dosage gradually increased until a maintenance level is reached, which is then continued for a minimum of three years.

Although subcutaneous immunotherapy is widely accepted, concerns about the risk of anaphylaxis have led to the development of alternative methods such as oral, sublingual, and nasal administration. Among these, sublingual immunotherapy (SLIT) has been widely used across Europe for the past two decades, favored for its safety, non-invasive delivery, and convenience of home use, making it a preferred choice over injections.

Research shows that immunotherapy is effective in both children and adults with AR caused by pollen and dust mites. It also helps prevent the progression to asthma and reduces the likelihood of developing new allergic sensitivities. Notably,

the benefits of immunotherapy, especially SLIT, are long-lasting even after treatment ends, highlighting its safety and efficacy (31).

Recent advances

Significant progress has been made in elucidating the pathophysiology of AR, particularly the complex interactions between genetic predisposition, environmental factors, and immune dysregulation. The importance of the epithelial barrier and the microbiome in the development of AR has gained attention, identifying new therapeutic targets.

Innovations in diagnostic techniques, such as component-resolved diagnostics and molecular allergology, have improved the precision of allergen identification, leading to more individualized treatment approaches. In treatment, novel biologics targeting specific pathways involved in AR, such as monoclonal antibodies against immunoglobulin E (IgE) and interleukin (IL)-4/13, have shown promise in alleviating symptoms in patients with moderate to severe AR. There is also a renewed emphasis on non-pharmacological strategies, including allergen avoidance and immunotherapy, providing holistic management options. Additionally, the incorporation of digital health tools and mobile applications has empowered patients to track symptoms and optimize treatment plans. The article advocates for a multidisciplinary approach to AR management, emphasizing patient-centered care, and outlines the need for ongoing research to continue advancing AR treatment and improve the quality of life for affected individuals. [(36)]

Bronchial Asthma

Definition

"Bronchial asthma is defined as a chronic inflammatory disease of airways characterized by bronchial hyperreactivity and a variable degree of airway obstruction (Ukena D et al)"[37]

Bronchial Asthma-Overview

Asthma is a common but complex condition seen in up to one third of people around the world and is marked by ongoing inflammation of the airways.[38] It is identified by recurring respiratory symptoms such as wheezing, breathlessness, chest congestion, and coughing, which fluctuate in severity and frequency. This disease often involves increased sensitivity of the airways and inflammation, leading to inconsistent restriction of airflow during exhalation. A combination of demographic, clinical, and pathophysiological data related to asthma are commonly clubbed under the heading 'asthma phenotypes'.[39] For patients with severe asthma, treatments tailored to specific phenotypes are available. However, aside from severe cases, there is no strong correlation between distinct pathological features and specific clinical patterns or responses to treatment. These asthma phenotypes commonly used in practice include:

Allergic asthma

This is the most common and the point of discussion in this study phenotype often starting in childhood and gradually becoming more symptomatic as age increases. Mostly likely it is linked to a history of allergic conditions such as eczema, allergic rhinitis, or food and drug allergies which may sometimes occur in

combination. They generally respond well to the initial treatment if begun at an early period of disease presentation. Sputum samples of these patients when collected before introducing any treatment protocol will show an eosinophilic predominant picture that is consistent with the etiology.

Non-allergic asthma

In some individuals, asthma is not linked to allergies. The sputum cellular profile in these patients may show a mixed picture which is neutrophilic, eosinophilic, or paucigranulocytic. These patients often show a reduced short-term response to initial therapy.

Adult-onset (late-onset) asthma

Sometimes adults develop symptoms of asthma even though they have no significant background contributing to the disease. These patients are often nonallergic and may require higher doses or may be less responsive to corticosteroid treatment. One important cause to be ruled out is, occupational asthma, due to workplace exposures in these patients.

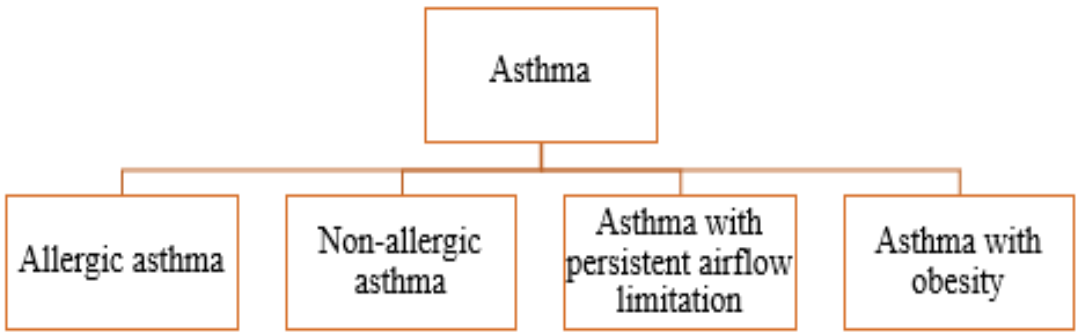

Figure 6: Types of asthma

Asthma with persistent airflow limitation

Some long-term asthma patients develop persistent or incompletely reversible airflow limitation, likely due to airway wall remodelling.

Asthma with obesity

Sometimes obese individuals exhibit asthmatic features with significant respiratory symptoms and should be treated with caution considering the etiopathogenesis.

Pathophysiology of Bronchial asthma

Airflow limitation in asthma is recurrent and results from various changes within the airways. These changes include:

Bronchoconstriction

The primary physiological event in asthma leading to clinical symptoms is airway narrowing, which hampers airflow. During acute asthma attacks, bronchial smooth muscle contraction occurs rapidly in response to various stimuli, such as allergens and irritants. When allergens trigger bronchoconstriction, it is primarily due to an IgE-mediated release of mediators from mast cells, including histamine, tryptase, leukotrienes, and prostaglandins, which directly contract smooth muscle in the airways. Additionally, some patients may experience acute airflow obstruction due to aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs), which also involve mediator release from airway cells. Other stimuli like exercise, cold air exposure, and irritants can lead to acute airflow obstruction, although the specific mechanisms regulating these responses are less clearly understood and appear to correlate with underlying airway inflammation. Stress may also be a contributing

factor in triggering asthma exacerbations, potentially through the enhanced production of pro-inflammatory cytokines.

Airway Edema

As asthma persists and inflammation progresses, other factors further restrict airflow, including edema, inflammation, mucus hyper-secretion, and the formation of thick mucus plugs. Structural changes in the airways, such as hypertrophy and hyperplasia of smooth muscle, may also develop and might not fully respond to standard treatments.

Airway Hyper-responsiveness

Airway hyper-responsiveness refers to an exaggerated bronchoconstrictor response to various stimuli, which is a significant characteristic of asthma. The severity of hyper-responsiveness, often measured with methacholine challenge tests, correlates with clinical asthma severity. The underlying mechanisms are multifaceted, involving inflammation, dysfunctional neuroregulation, and structural alterations; however, inflammation is a key contributor. Targeting inflammation can help decrease airway hyper-responsiveness and improve asthma management.

Airway Remodeling

In some patients with asthma, airflow limitation may not be entirely reversible. Permanent structural changes can occur within the airways, leading to progressive loss of lung function that current therapies do not fully prevent or reverse. Airway remodeling includes the activation of structural cells, causing permanent alterations in the airway that exacerbate airflow obstruction and responsiveness, ultimately making patients less responsive to treatment. These structural changes can

encompass thickening of the sub-basement membrane, subepithelial fibrosis, hypertrophy and hyperplasia of airway smooth muscle, and increased mucous gland activity. The regulation of repair and remodeling processes remains poorly defined, yet they are believed to play a critical role in the chronicity of the disease and the limitations in therapeutic responses.

Pathophysiologic mechanisms in the development of airway inflammation

Inflammation plays a central role in the pathophysiology of asthma, characterized by the interaction of various cell types and mediators that lead to bronchial inflammation and airflow limitations. Symptoms like coughing, wheezing, and shortness of breath are common, and although distinct asthma phenotypes exist, the presence of airway inflammation remains consistent across different severities and durations of the disease. Recently, advances in the understanding of innate immune responses, particularly regarding lymphocytes, have shed light on asthma's complexity. Increased activity of T helper 2 (Th2) cells has been linked to eosinophilic inflammation, which is a hallmark of asthma. As these cells produce cytokines such as IL-4, IL-5, and IL-13, they also contribute to IgE overproduction and airway hyperresponsiveness.

The role of mast cells is significant, as their activation leads to the release of bronchoconstrictive mediators like histamine and leukotrienes, contributing to airway constriction. Eosinophils are frequently found in the airways of asthmatic patients and correlate with the severity of the condition. While therapies reducing eosinophil levels through corticosteroids have shown effectiveness, their exclusive role in asthma management is being re-evaluated. Neutrophils, on the other hand, are more prevalent

in severe asthma cases and during acute exacerbations, although their exact contribution remains uncertain.

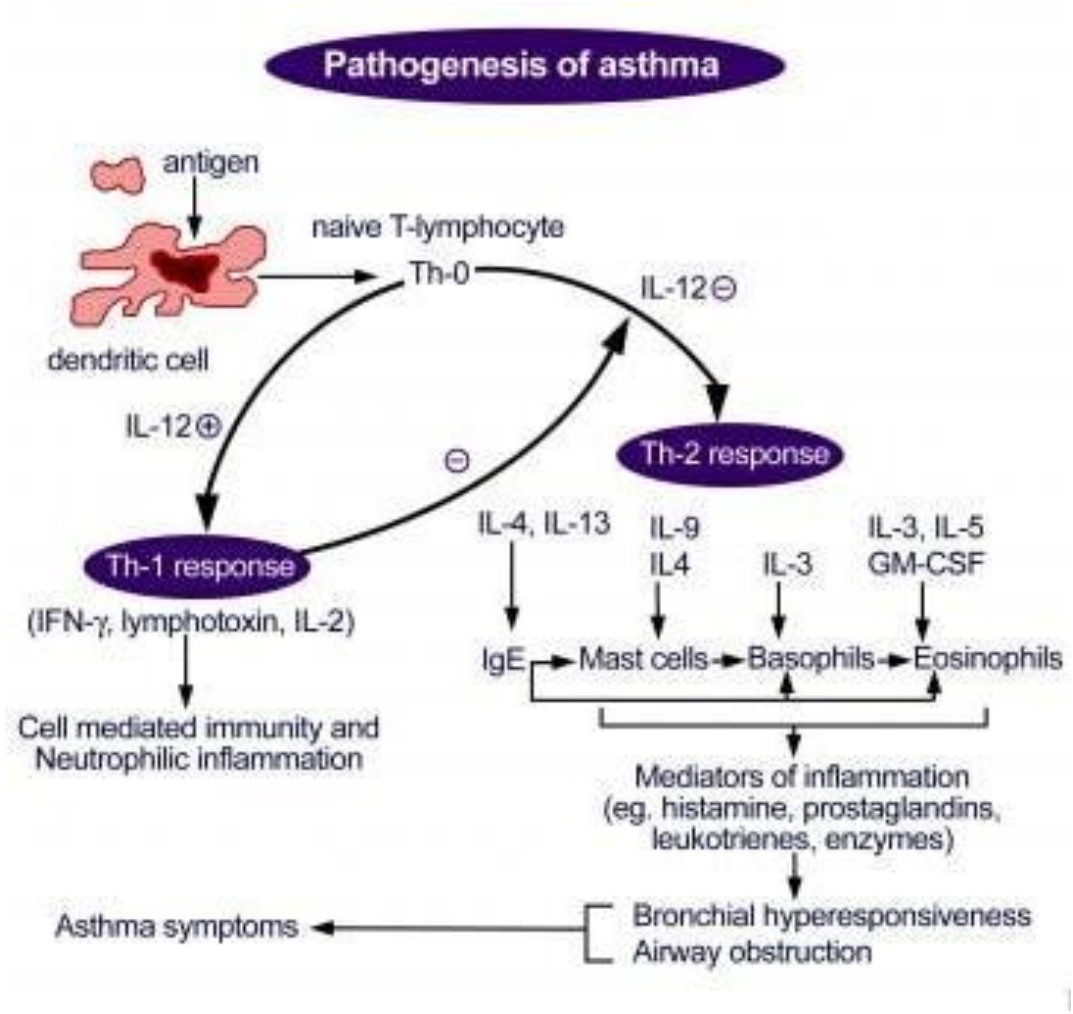

Figure 7: Pathogenesis of asthma

Dendritic cells serve as crucial antigen-presenting cells, interacting with airway allergens and activating T cells, while macrophages represent a significant population involved in the inflammatory response. Overall, understanding these cellular mechanisms is vital for advancing asthma treatment and management strategies.

Pathogenesis of bronchial asthma

The initiation of the inflammatory process in asthma and the reasons behind individual susceptibility are areas of active research. Although there is no definitive explanation, emerging evidence suggests that the development of asthma primarily occurs early in life. The expression of asthma is a complex interaction between host factors, particularly genetic predispositions, and environmental exposures during critical stages of immune system development.

Host Factors:

Research has highlighted the roles of both innate and adaptive immune responses in regulating inflammation. A significant focus has been on the imbalance between T helper 1 (Th1) and T helper 2 (Th2) cytokine profiles. Asthma is often characterized by a predominance of Th2 cytokines, which mediate allergic inflammation. The "hygiene hypothesis" posits that the immune system of infants is skewed toward Th2 responses, and exposure to infections can help balance Th1 and Th2 activity, reducing asthma incidence. Factors such as certain infections, presence of older siblings, and reduced antibiotic use have been associated with lower asthma rates. In contrast, a lack of these experiences can lead to persistent Th2 patterns, promoting IgE production in response to environmental allergens.

Genetics:

Asthma has a recognized genetic component, although the genes involved in its development present a complex and not fully understood picture. Numerous genes have been implicated in asthma's presence and characteristics, influencing IgE production, airway hyperresponsiveness, and the regulation of inflammatory

mediators. Ongoing studies are also examining genetic variations that may affect therapy responses, particularly regarding polymorphisms in beta-adrenergic and corticosteroid receptors.

Sex:

The prevalence of asthma is higher in boys during early childhood; however, after puberty, the ratio shifts, with women becoming more frequently affected. This change raises questions about the specific impacts of sex and sex hormones on asthma development and manifestation.

The multifactorial nature of asthma, suggest that both genetic predispositions and environmental interactions play crucial roles in the disease's onset and progression. [40]

Diagnosis of asthma

Features suggestive of a confirmed variable expiratory airflow limitation point towards the diagnosis of asthma. These include fluctuating expiratory airflow restriction, meaning the ability to exhale changes over time and to a greater degree than in healthy individuals. In asthma, lung function can range from entirely normal to severely limited within the same person. When asthma is poorly managed, these fluctuations in lung function tend to be more pronounced compared to well-managed asthma.[41] A decreased forced expiratory volume in one second (FEV1) may be seen in a number of clinical conditions but a low forced expiratory volume in one second and forced vital capacity (FEV1/FVC) ratio suggests an expiratory airflow limitation. Increase in FEV1 from baseline of >12%, detected 10–15 minutes after 200–400 mcg of bronchodilator like salbutamol or its equivalent, is touted as a positive bronchodilator response that points towards an asthma diagnosis.

Other confirmatory methods include a diurnal variability of Peak expiratory flow rate >13% and a positive exercise and bronchial challenge test. Certain bronchial provocation tests for airway hyper responsiveness measurement are available that utilise a variety of agents. Allergy tests, mainly the skin prick tests are also widely seen and newer tests of serum IgE detection as in this current study also help tilt towards the diagnosis of asthma.

Treatment for asthma

The initial approach to treating pediatric asthma involves evaluating the severity and intensity of symptoms, as well as the risk of future exacerbations. For children aged 5 and under, the potential for developing persistent asthma is also taken into account. This evaluation includes reviewing how often daytime and night time symptoms occur, managing those symptoms with short-acting β-agonists (SABAs), assessing how symptoms affect daily activities, and performing spirometry tests for children aged 5 and older. Additionally, the number of exacerbations that required glucocorticoid treatment in the previous year helps assess the likelihood of future episodes.

Instead of solely determining severity, healthcare providers evaluate how well patients are controlling their symptoms while on controller therapy. Experts recommend incorporating spirometry results with symptom assessments and medication usage to effectively measure asthma control. If there is uncertainty regarding a patient's diagnosis or management level, measuring fractional exhaled nitric oxide (FENO) can be beneficial.

Non-Pharmacological Management

Non-pharmacological management strategies for asthma emphasize patient education. Personalized one-on-one education provided by primary clinicians, which has been shown to reduce asthma exacerbations and hospital visits. Healthcare providers should offer culturally relevant asthma education that covers the condition, its symptoms, individual triggers, and strategies for avoidance. Preventing exposure to environmental triggers, including both firsthand and secondhand tobacco smoke, food or medication allergens, and various pollutants, is critical.

Patients and their caregivers need to understand the correct use of inhalers and differentiate between rescue, controller, and combination medications. Clinicians should also address any barriers to medication adherence and collaborate with patients to resolve any concerns, thereby improving compliance.

Pharmacological Management

The study outlines a stepwise approach to pharmacological therapy for children aged 5 and younger as per the guidelines provided by the Global Initiative for Asthma (GINA):

Step 1: All children with wheezing should have access to a SABA, except infants under 1 year of age who wheeze due to bronchiolitis. If a SABA is required more than twice a week for a month, the patient should move to Step 2.

Step 2: Healthcare providers should introduce a low-dose inhaled corticosteroid (ICS) to be taken daily along with as-needed SABA, maintaining this regimen for a minimum of 3 months. Persistent poorly controlled symptoms necessitate confirming the absence of alternative diagnoses, ensuring medication compliance, checking

inhaler technique, and assessing exposure to tobacco smoke or environmental allergens.

Step 3: If symptoms continue, increasing the ICS dose for three months is recommended. Should the symptoms remain unmanageable, a referral to a specialist in asthma care is advised.

Step 4: At this stage, further management options include increasing the ICS dose, adding a leukotriene receptor antagonist (LTRA), combining long-acting β-agonists (LABA) with the ICS, or temporarily prescribing a low-dose oral corticosteroid (OCS) until symptoms improve.[42]

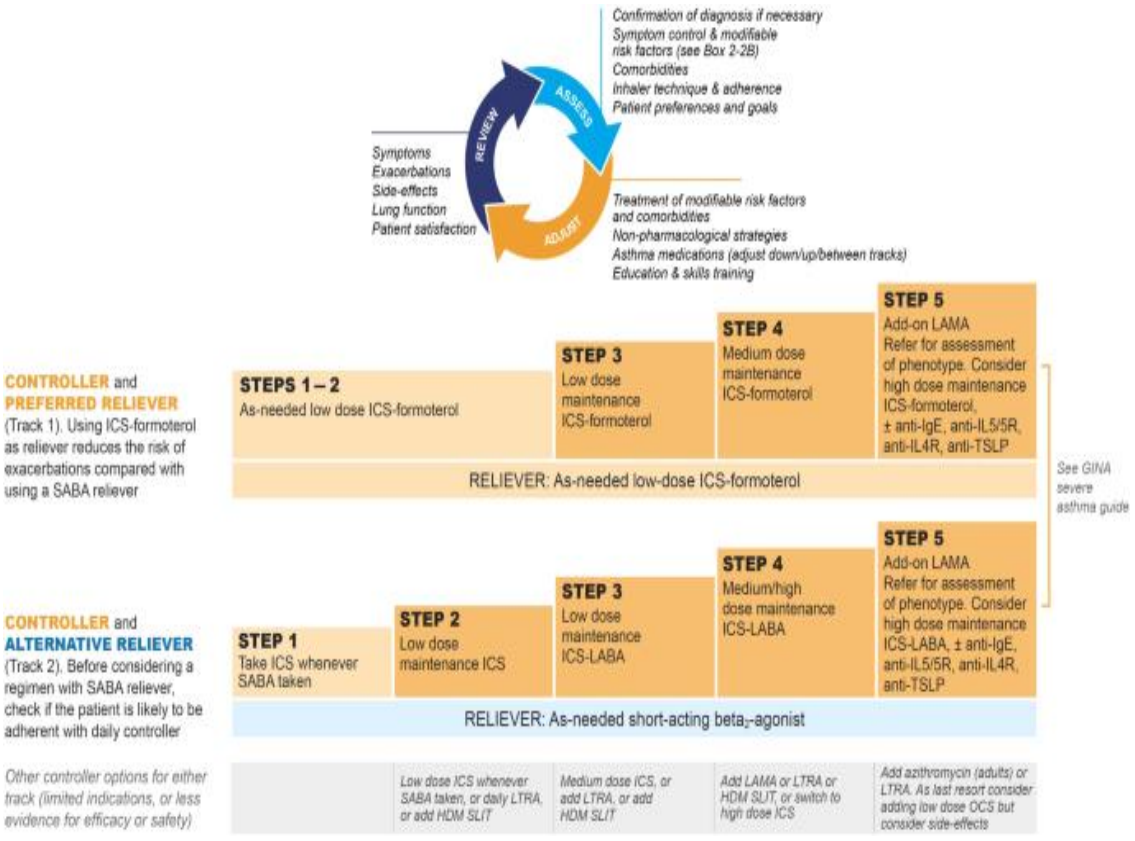

Figure 8: Recommendations from Global Initiative for Asthma (GINA) 2022

United Airway Disease

Definition

United airway disease is characterized by inflammation of respiratory tract in which asthma and rhinitis are the upper and lower respiratory tract manifestations, respectively, of the same disease process. Irrespective of the cause, the upper and lower respiratory tract manifestations are characterized by a systemic inflammatory response. Individuals with rhinitis or asthma should be assessed for the co-existence of other disease. [(43)]

Burden of disease -The global prevalence of united airway disease was 10 to 40%. [(44)]

United airway disease-Overview

United airway disease is considered as a single inflammatory process of the respiratory tract involving both upper respiratory tract and lower respiratory tract. The concept of united airway disease was introduced in the early 2000s.[(45)]

Each segment of the respiratory system has specialized functions: the nose filters, humidifiers, and warm incoming air while detecting odours; the larynx facilitates phonation and the lungs are responsible for the gas exchange. However, respiratory conditions often affect both the upper and lower respiratory tracts simultaneously. For example, asthma and rhinitis frequently coexists, with upper respiratory tract infections capable of worsening asthma. Additionally, rhinitis is recognized as an independent risk factor for development of asthma.

The respiratory tract is a continuous system with shared anatomical and histological characteristics primarily designed for air passage. Both upper and lower airways exhibit a similar vulnerability to allergens, infections, environmental pollutants, and certain medications, responding to these agents in comparable ways. The entire tract is lined with ciliated epithelium, mucinous glands and a dense vascular and nervous network, suggesting a strong connection between nasal and bronchial functions. This supports the theory of a sino-nasal-bronchial reflex, which may contribute to common pathogenic mechanisms in respiratory diseases.

Allergic conditions often begin in the upper respiratory tract and progress to the lower airways over time. Individuals with allergies experience widespread inflammation affecting the nose, sinuses and bronchi. Localized nasal inflammation can have systemic consequences through soluble mediators, influencing bone marrow activity, and increasing the production of eosinophil precursors in response to nasal allergen exposure. Furthermore, allergic reactions in the nasal passages can trigger pulmonary inflammation, even in individuals without a prior history of asthma or bronchial hyper-responsiveness (BHR).

Bronchial allergen exposure can induce nasal inflammation and symptoms within 24 hours in patients with allergic rhinitis. Conversely, nasal allergen challenges may result in the respiratory function within 4 to 6 hours. This highlights the interconnected nature of the respiratory system, leading to the concept of united airway disease, a recognition that the respiratory tract functions as a single, integrated entity both anatomically and physiologically.[11]

Pathophysiology

This mainly presents itself in two forms-allergic (atopic or exogenous) and non-allergic (nonatopic or endogenous). The allergic phenotype predominates in most children and over 50% of adults, wherein the condition is linked to sensitization to allergens and the presence of specific Immunoglobulin E (IgE) antibodies in the serum.[46] The spectrum of allergic airway disease stems most likely due to the presence of many allergens in the surroundings of which the most common ones are being included as a part of the study Dust mite allergy, a common affliction triggered by allergens residing in household dust, manifests predominantly during childhood, with symptoms typically emerging before the age of 20. It was first identified as an allergen around 1920 and then in 1967, it was identified as the cause for house dust allergy by Voorhorst et al.[47] This allergic condition is widespread, affecting a substantial portion, estimated between 40% to 85% of individuals grappling with asthma and allergies across regions like America, Europe, Southeast Asia and Australia. The digestive enzyme, peptidase 1, abundantly present in dust mite fecal matter, serves as a potent allergen inducer, contributing significantly to the onset of this hypersensitivity reaction. CD4+ cells prompt B cells to generate IgE antibodies specific to dust mite allergens which attach to mast cells and basophils and on re-exposure to these allergens, cross-linking of IgE-bound cells occurs, leading to degranulation and the release of inflammatory mediators, resulting in allergic symptoms like rhinitis and asthma.

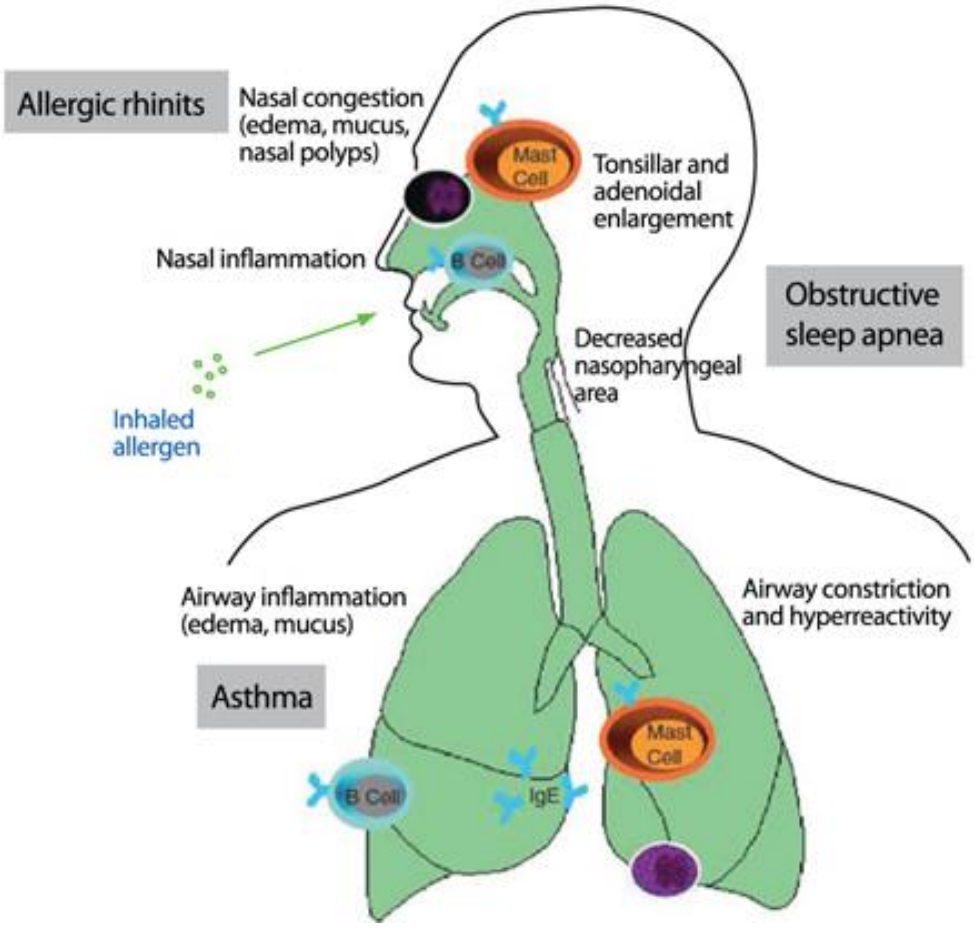

Figure 9: Pathophysiology of allergic rhinitis and asthma

Diagnosis of united airway disease

Clinical history, physical examination, imaging like computer tomography (CT) and magnetic resonance imaging (MRI), allergy and laboratory investigations, pulmonary function test (PFT), and tissue histopathology are used in diagnosis of united airway disease.[48]

Treatment of united airway disease

Effective management of unified airway disease, encompasses both upper and lower airway conditions involve targeted therapies which addresses inflammation across the entire respiratory tract. Treatment of allergic rhinitis with intranasal

corticosteroid sprays alleviates nasal symptoms and also improves asthma control, reducing both symptoms and objective pulmonary function impairment. Similarly, oral antihistamines and leukotriene receptor antagonists have shown comparable benefits in managing both nasal and bronchial inflammation.

Beyond symptom relief, these treatments have a significant impact on patient outcomes at a broader level. Patients with both allergic rhinitis and asthma who receive appropriate treatment for their nasal condition experience fewer asthma-related hospitalizations and emergency room visits compared to those who do not receive or adhere to treatment.

Managing chronic sinus disease has also been linked to better asthma control. Effective management of sinusitis reduces the need for asthma medications, enhances pulmonary functions and decreases the frequency of asthma exacerbations. This highlights the interconnected nature of upper and lower airway inflammation and underscores the importance of comprehensive treatment strategies.

Another crucial intervention in UAD management is immunotherapy, which has positive effects on both upper and lower airway conditions. By modulating the immune response, immunotherapy not only reduces allergic rhinitis symptoms, but also contributes to improved asthma control, reinforcing the idea that a systemic approach to airway inflammation can yield widespread benefits.

Proper therapeutic interventions targeting both upper and lower airways can enhance patient outcomes, reduce healthcare utilization and improve quality of life for individuals suffering from united airway diseases. [(49)]

Tests for various allergic airway diseases in practice

Skin prick test (SPT)

The skin prick test is the most widely used method for diagnosing allergies due to its reliability, safety, convenience, and low cost. It involves pricking skin, usually on the forearm, with a lancet containing an allergen extract, allowing multiple allergens to be tested in 15 to 20 minutes. SPT is less distressing for children compared to venipuncture and correlates well (85% to 95%) with invitro tests for specific IgE measurement. Despite its high positive predictive value (95% to 100%), results must be interpreted alongside clinical history, as sensitization may not equal to clinical allergy. Although systemic reactions are rare, invitro IgE tests are recommended if the patient has had previous severe reactions. Skin prick test is commonly used to identify inhalant allergies related to type I, IgE-mediated hypersensitivity. [19]

Intradermal testing

Intradermal testing is usually conducted if SPT results are negative to detect low-sensitivity allergens. Some clinicians routinely perform ID testing for all allergens that tested negative on SPT, while others only do so for allergens with a high suspicion of clinical relevance. A negative intradermal test following a negative SPT suggests the patients is unlikely to be sensitive to that allergen. Conversely, a positive intradermal test after a negative SPT may indicate a clinically significant allergen. Additional intradermal testing may be warranted in patients with a high suspicion of inhalant allergies, especially for mold or unusual inhalant exposures. [50]

Nasal allergen provocation test (NAPT)

NAPT is a method used to simulate the upper airway's response to natural allergen or irritant exposure and aids in the differential diagnosis of rhinitis conditions. It boasts high specificity (83.7%) and sensitivity (100%). Through the NAPT, diagnosis can be made for allergic rhinitis (40.2%), non-allergic rhinitis (38.8%), and local allergic reactions. Nasal allergen provocation test is a clinical procedure designed to evaluate the response of upper airways to exposure to specific allergens or irritants. It serves both diagnostic and research purposes, allowing conditions by assessing the biochemical mediators involved in allergic reactions. NAPT is particularly valuable in differentiating between various types of rhinitis, including allergic rhinitis, and non-allergic rhinitis. [51]

ImmunoCAP

ImmunoCAP is a highly standardized in vitro diagnostic system used to quantitatively measure allergen-specific IgE antibodies in human serum or plasma. It is considered the gold standard in molecular allergy diagnostics and is widely used in both clinical and research settings due to its high sensitivity, reproducibility and standardization.

There are two types of ImmunoCAP assays. One singleplex ImmunoCAP, which measures IgE against one specific allergen per test and is suitable for targeted testing and routine screening. Another, multiplex ImmunoCAP ISAC (Immuno Solid-phase allergen chip) simultaneously measures reactivity to 112 allergen components derived from over 50 allergen sources using microarray technology. Useful in complex cases or for molecular phenotyping.

The key features of ImmunoCAP is it is based on fluorescence enzyme immunoassay principle. It uses allergen extracts or purified allergen molecules immobilized on a solid cellulose-based matrix. It has a high analytical sensitivity with a wide measurable range (0.1-100 kUA/L).

This test is preferred over SPT especially in paediatric, multi-sensitized or food allergy patients as it is non-invasive, unaffected by medications, objective, quantitative, reproducible, and ideal for use in both primary and tertiary care settings.

Difference between EAST and ImmunoCAP

EAST and ImmunoCAP are both in vitro methods used to detecting allergen-specific IgE antibodies in the blood. EAST is based on enzyme-linked detection and is commonly used in many laboratories, whereas, ImmunoCAP is a newer, more advanced platform with higher sensitivity, standardization and reproducibility.

ImmunoCAP provides quantitative results traceable to WHO standards and allows for component-resolved diagnostics using either single allergens (singleplex) or multiple allergen molecules (multiplex using ImmunoCAP ISAC). Unlike EAST, ImmunoCAP provides better precision, automated and is less prone to interference from other antibodies like IgG. [52, 53]

Enzyme Allergo Sorbent Test (EAST)

Development of EAST

Two research teams independently developed the direct enzyme-linked immunosorbent assay (ELISA): Engvall and Perlman, and Van Weemen and Schuurs. The ELISA emerged as an enhancement of the radioimmunoassay (RIA), achieved by tagging antigens and antibodies with enzymes instead of using radioactive iodine-125.

The method was initially utilized to measure IgG levels in rabbit serum and later quantified human chorionic gonadotropin in urine using horseradish peroxidase, both within the same year. Since its introduction, ELISA has been widely adopted for various applications and has become a standard technique in laboratory research and diagnostics globally.

The original ELISA method utilized chromogenic reporter molecules and substrates to create a noticeable color change, allowing the detection of antigens. Advancements in the technique have led to the use of fluorogenic, quantitative PCR, and electrochemiluminescent reporters to generate signals. Some newer methods do not depend on enzyme-linked substrates, instead using non-enzymatic reporters while still following the ELISA principle.

The most recent innovation, introduced in 2012, involved an ultra-sensitive enzyme-based ELISA that employs nanoparticles as chromogenic reporters. This technique provides a color signal that can be seen without special equipment, with blue indicating a positive result and red indicating a negative one. However, this method is qualitative, meaning it can only confirm the presence or absence of an analyte rather than measure its concentration.[54]

EAST

Enzyme allegro sorbent test is an immunological assay utilized for the quantification of specific IgE antibodies in serum, specifically in relation to allergens such as food and inhalant substances. This test is particularly valuable in the diagnosis of allergic conditions, allowing for the identification of sensitization to a range of allergens. EAST operates on the principle of enzyme-linked immunosorbent assay (ELISA), where allergen extracts are immobilized on test strips. Each strips typically

contains parallel lines made up of various allergen extracts, facilitating the simultaneous testing of multiple allergens. When a patient's serum containing IgE antibodies is applied to the strip, specific antibody-antigen interactions occur. Following this, a secondary enzyme-labelled antibody binds to the IgE, enabling detection through a substrate reaction that produce a measurable colour change. The intensity of colour bands is quantitatively analyzed using digital evaluation systems, such as the EURO line scan, which provides precise measurements of the IgE levels. Any score above a specific threshold, usually set at 1, is interpretated as a positive result, indicating sensitization to the corresponding allergens. EAST is advantageous due to its ability to assess multiple sensitivities in a single test, reducing the need for extensive skin testing, and increasing patient comfort. Moreover, it plays a critical role in differentiating between allergic and non-allergic responses, guiding clinicians in developing targeted management strategies for allergic patients. The test has robust specificity and sensitivity, making it a reliable tool in both clinical diagnostics and research settings related to allergies. Overall, EAST is a useful diagnostic tool that enhances allergen identification and contribution to a better understanding of allergic diseases by quantitatively measuring IgE antibodies against a wide array of allergens. (18)

Advantages and disadvantages of EAST

Table 2: Advantages and disadvantages of EAST

Advantages	**Disadvantages**
High sensitivity and specificity	Less sensitive than SPT
Non-invasive and safer than skin test	Expensive and time consuming
Standardized and reproducible	Cannot differentiate between sensitization and clinical allergy
Detects a wide range of allergens	Delayed results
Useful for severe allergen cases	Limited accessibility in some regions

Comparison of SPT and EAST test

Table 3: Comparison of SPT and EAST Test

TEST	**SPT**	**EAST**
Methodology	Involves applying a small amount of allergen extract to the skin, usually on the forearm or back, and then using a lancet to prick the skin's surface, Reaction is observed after 15-20 minutes for development of wheals and erythema, indicating a positive allergic response.	An invitro blood test that quantifies specific IgE antibodies against various allergens present in the serum. Utilizes test strips coated with allergen extracts, and the intensity of the resulting bands is measured through an enzyme-linked reaction, indicating the amount of IgE present.
Speed	Faster than EAST. 15 minutes	Slower than SPT. Few days to 2 weeks
Cost	Less expensive	Expensive than SPT
Types of allergen tested	Generally used for common inhalants (Eg: pollen, dust, mites) and foods. Tests multiple allergens simultaneously.	Capable of testing broader range of allergen, including uncommon ones. Allows for simultaneous detection of multiple allergen-specific IgE in a single test
Sensitivity and specificity	High sensitivity and specificity More changes of false positives Sensitivity: 68-100% Specificity: 70-97%	High sensitivity and specificity Less false positivity Sensitivity: 62% Specificity: 96%
Invasive	Minor invasive procedure	Blood test, invasive
Results	Determined based on the size of wheals, indicating a stronger allergic response. Immediate results can lead to instant clinical decisions.	Results quantified based on the level of specific IgE detected, with scores representing the extent of sensitization. Requires careful interpretation of IgE levels in conjunction with clinical history to confirm allergy, as sensitization does not always correlate with clinical symptoms.

Treatment

The long-term objectives of asthma treatment are to maintain effective control over symptoms and reduce the future risk of asthma-related complications, such as death, flare-ups, chronic breathing difficulties, and treatment side effects. It's also important to understand and incorporate the patient's personal goals regarding asthma and treatment plan. For people with infrequent asthma symptoms (less than twice a month and no major risks for flare-ups), the typical recommendation is to use a quick-relief inhaler short acting beta agonist (SABA) as needed. For those with asthma symptoms or the need for a reliever inhaler at least twice a month but less than daily, the preferred treatment is a low-dose inhaled steroid (ICS) taken every day, along with a quick-relief inhaler (SABA) as needed. It's important to consider whether the person will stick to a regular treatment plan if they're only using the quick-relief inhaler. If someone has troublesome asthma symptoms on most days (like 4–5 days a week), or if they wake up due to asthma once a week or more, especially if they have risk factors for flare-ups, the treatment options include a low-dose ICS combined with a long-acting bronchodilator (LABA) plus a quick-relief inhaler as needed, or a medium-dose ICS plus a quick-relief inhaler. Another choice is to use a very-low-dose ICS-formoterol for both daily control and as a reliever. For someone experiencing severely uncontrolled asthma or a sudden worsening of symptoms, the treatment should begin with regular use of a medium-dose ICS-LABA, along with a quick relief inhaler.

For prevention of these allergic airway diseases, patients should avoid medications that might worsen symptoms, especially NSAIDs, unless no previous reactions occurred. A healthy diet with plenty of fruits and vegetables is beneficial, and weight reduction is recommended for obese patients, especially with regular

exercise. Indoor air should be clean and allergen exposure minimized where possible, especially in damp or moldy spaces. During high pollen seasons, closing windows and staying indoors may help. Breathing exercises and relaxation techniques can ease symptoms but won't reduce flare-ups. Addressing social and emotional factors, including mental health support, is key to reducing asthma-related stress and hospital visits.

Comparison of allergy in India and western countries

Prevalence of asthma in children in India ranges from 2 to 18.2%. Prevalence of allergic rhinitis in children between age 6-7 years was reported as 11.3% and between the age 13-14 years was reported as 24.4%. [(55)] Prevalence of coexistence of both asthma and allergic rhinitis was reported up to 65.24%.[(56)] In USA, the prevalence of asthma ranges from 23-39%. The prevalence of allergic rhinitis at 6-7 years and 13-14 years group were 29.4% and 46.2% respectively. [(57)]

House dust mites are the predominant aeroallergens in India. In a study from South India, 41% of the patients with allergic rhinitis and asthma were sensitized to house dust mites with Dermatophagoides pteronyssinus (36.9%) and Dermatophagoides farinae (22.4%) being most common.[(58)] Among sensitized children, 54% reacted to dust mite mix and 44% reacted to tree pollens in a study conducted in USA. Hence, common aeroallergen in USA in children were house dust mites and tree pollen. [(59)]

Urbanization and lifestyle changes such as increased exposure to air pollution and changes in housing have contributed to rising allergy rates among children in India. Socioeconomic status influences access to healthcare and awareness of allergies, often leading to underdiagnosis in India.[(60)]

Socioeconomic status affect healthcare accessibility and quality. Disparities exist in allergen exposure and treatment, particularly in marginalized communities. Increased awareness and effective healthcare services are generally better established in the USA, leading to higher diagnostic rates. [61]

Counseling the parents for allergy testing in children

Allergy testing in children is essential because allergic diseases are common in childhood and can lead to significant health issues and reduced quality of life for both the child and their family. Proper allergy testing is crucial for effective management, including identifying allergens to avoid, guiding medication use, and determining the need for immunotherapy. It helps pinpoint specific allergic triggers, even in young children, especially when symptoms are persistent, recurrent, or severe. Early and accurate diagnosis ensures targeted treatment and better long-term outcomes. [61]

When counselling parents about allergy testing for their children, it's important to clearly communicate the purpose and benefits of identifying specific allergens. Helping them understand that pinpointing the triggers can significantly improve symptom control and quality of life by allowing for targeted management strategies. Walking them through the testing process step by step, for tests like skin prick test or blood test is crucial, so they know what to expect and can feel confident in the procedure when the child takes it.

It is essential to be sensitive to any concerns they may have about potential reactions during testing, and reassuring them about the safety measures in place is important. After allergens are identified for the children, providing clear and practical guidance on how to manage their child’s allergies is the next step. This includes strategies for avoiding known allergens, understanding food labelling to prevent

accidental exposure, and ensuring that emergency medications like epinephrine autoinjectors are always readily available and used correctly.

Encourage consistent follow-up with an allergist to monitor the child's condition and adjust the care plan as needed. Finally, recommend support resources—such as allergy education programs or parent support groups—to help them navigate their child's condition with confidence and community support.[62]

Literature from previous studies

1. A study conducted by Umanets TR et al., 2020 aimed to study the sensitization profile of children with asthma respiratory diseases. Children between the age of 5 to 17 with allergic respiratory diseases were included in this study with 51 children with allergic rhinitis, 31 children with allergic rhinitis combined with asthma and 11 children with isolated asthma. All the study participants underwent the molecular diagnostic test ALEX. Cat allergen Fel d 1 was common across all groups. Specific allergens were associated with particular conditions: house dust mites in AR with asthma, mold and grass allergens in isolated AR, and dog and wasp allergens in isolated asthma. Polysensitized children exhibited diverse allergen sensitivities, including PR-10 molecules, with unique patterns depending on the condition. This highlights the complexity of allergen sensitization in these children.[63]
2. A study conducted by Feliu A et al., 2013 aimed to identify the primary causes of allergies in paediatric patients aged 14 and below living in areas with high allergen concentrations and to evaluate the changes in sensitization profiles after three years. The research involved 187 children presenting with symptoms of allergic diseases such as rhino-conjunctivitis and asthma, across

five allergy units. Diagnosis was confirmed through clinical history and allergen testing, including specific IgE measurements for major allergens. Findings revealed that patients were sensitized to both seasonal allergens (notably grass, olive, cypress and Cynodont dactylon) and perennial allergens (like Alternaria alternata) as well as common panallergens like profilin and lipid transfer protein. Almost 60% of the study participants showed polysensitization, with age-related increases in sensitization to specific major allergens (eg: Cup s1, Phl p1, Sal k1). Additionally, patients sensitized to profilin exhibited a greater number of sensitizations compared to those who were not. The study concludes that many paediatric patients in high-exposure areas are polysensitized, with significant sensitization to panallergens, highlighting the need for advanced diagnostic methods like component-resolved diagnosis for better management of allergic conditions. [(64)]

3. The study conducted by Dai L et al., 2020 aimed to evaluate sensitization profile to aeroallergens in children with allergic rhinitis and asthma, utilizing skin prick test and to investigate differences in sensitization based on allergic disease type, sex and age. A total of 230 hospitalized children with allergic rhinitis and/ or asthma were included in this retrospective study from June 2017 to September 2019. The results indicated that 67.4% of study participants tested positive for skin prick test with the most common allergen being Dermatophagoides pteronyssinus (59.3%), Dermatophagoides farina (58.7%), Blomia tropicalis (40.3%), dog hair (36.1%) and Blattella germanica (20.4%). Notably, over 90% of the children were sensitized to two or more allergens. Adolescents showed a higher frequency of sensitization to Blomia tropicalis, dog hair and multiple allergens (≥5) compared to younger children. Furthermore, boys displayed a significantly higher positivity rate for skin prick

test and double-allergen sensitization rates compared to girls. Additionally, children with asthma and rhinitis exhibited increased sensitization rates to Dermatophagoides pteronyssinus and Dermatophagoides farina compared to those with only allergic rhinitis or asthma. In conclusion, the findings highlight that allergic sensitization are prevalent among children with allergic rhinitis and/ or asthma, with variations influenced by age, gender and co-occurrence of asthma and rhinitis.[65]

4. The study conducted by Raj D et al., aimed to assess the prevalence of sensitization to common aeroallergens in asthmatic children and to compare the characteristics of atopic and non-atopic individuals. The study was conducted at a paediatric chest clinic in a tertiary care centre in Northern India, and the study was conduced among children between 5 to 18 years diagnosed with asthma. Skin prick test was performed on 180 children. The results revealed that 100 children (55.6%) were sensitized to at least one aeroallergen, indicating atopy, and 68 (37.8%) children were sensitized to multiple allergens. The most prevalent sensitizations included housefly antigen (36.7%), rice grain dust (31.1%), cockroach (18.3%) and house dust mite antigen (7.8%). Atopic children showed significantly higher median fractional exhaled nitric oxide (FENO) levels during follow-up compared to non-atopic children (17.5 ppb Vs 13 ppb, p=0.002). Furthermore, a positive correlation was found between age and number of allergens to which children were sensitized (r=0.21, p=0.0049). In conclusion, more than half of the children with asthma in the study exhibited sensitization to one or more aeroallergens, with housefly and rice grain dust being the most common triggers. Atopic children demonstrated significantly elevated FENO levels compared to their non-atopic counterparts. [10]

5. A study conducted by Bhagyashree PR et al., 2023 aimed to document the prevalence of allergic sensitization among children with asthma between 5 to 18 years attending a tertiary care centre in Rajasthan, and to evaluate the impact of allergic sensitization on asthma control. A total of 85 children who were diagnosed with asthma were included in the study. A detailed history was taken and specific allergens were selected for the skin prick test, with a positive reaction defined as of 3mm or greater. Asthma control was assessed using Global Initiative for Asthma (GINA) guidelines and Asthma Control Questionnaire. Results revealed that 74% of the study participates tested positive for one or more allergens, with house dust mite being the most common allergen. However, there was no statistically significant relationship between the positive skin prick test results and asthma control. In conclusion, the study indicated that a significant proportion of children with asthma are sensitized to aeroallergens, predominantly to house dust mites. [(66)]
6. A study conducted by Kumar D et al., 2023 aimed to assess the prevalence of allergic disorders across all age groups and to identify common allergens responsible for various allergic manifestations including respiratory, gastrointestinal and skin-related in children aged 2 months to 18 years from northern India. It was conducted among patients with symptoms like asthma, allergic rhinitis, eczema, urticaria or gastrointestinal issues. The research includes skin prick test using allergens selected based on the history of the patient. A total of 458 patients were included in the study. The analysis from the skin prick test revelated that in patients with respiratory symptoms, the predominant allergens were house dust mites and cockroaches, followed by mosquitos. Those with gastrointestinal symptoms showed sensitization mainly to egg white, with soybean flour being the next most common allergen. Skin-

related allergic manifestations were most frequently associated with sensitization to house dust mite. Notable, house dust mite was also identified as a significant allergen in patients affected by both respiratory and skin conditions as well as those with respiratory and gastrointestinal issues, whereas groundnuts were linked to reactions in patients with concurrent gastrointestinal and skin symptoms. In conclusion, the study effectively identified the most common allergen affecting children in northern India with various allergic disorders, providing valuable insights for clinical management and treatment strategies.(67)

7. In a study conducted in 100 children by Chauhan K et al., in Lucknow, showed that allergen sensitization rates were high among asthmatic children, knowledge regarding common allergens could help alter the progression and achieve better asthma control in children and thus understanding the sensitization pattern in a local context is a necessary step to guide management.(68)
8. An analysis of serum IgE in 48 randomly selected children out of 86 between 5-17 years of age with asthma and other allergic respiratory illness was studied by Lazova S et al in children. Study suggested that establishing a spectrum of most common allergens is vital in asthma control and also to avoid allergen exposure and that these newer immunoblot techniques of determining serum IgE that are available in daily clinical and laboratory practices can be very helpful in guiding management.(69)
9. A large-scale study conducted in Shanghai examined allergen sensitization among nearly 40,000 children aged 1 month to 18 years with diagnosed allergic conditions. Testing for serum-specific IgE to 17 common allergens revealed that over 64% were sensitized to at least one. House and dust mites

were the most frequent airborne allergens, while milk and eggs were the top food allergens. Sensitization to airborne allergens increased with age, whereas food allergen sensitivity was more common in younger children, particularly between 1–3 years. Boys generally showed higher sensitization rates than girls. These findings highlight age and sex differences in allergen sensitivity and can inform targeted prevention and treatment strategies in the region, with potential applicability elsewhere. [70]

10. The study conducted by Yan et al examined allergen sensitization patterns among 7,996 adults aged 18–60 with allergic diseases in Shanghai, using skin prick test data. The most prevalent allergens were house dust mites, specifically *Dermatophagoides farinae* and *D. pteronyssinus,* along with *Blomia tropicalis*. Sensitization to cockroaches, pet dander, molds, and shrimp was also observed. Younger adults (18–30 years) were more reactive to mites and animal dander, while older individuals (above 40 years) showed higher sensitivity to cockroaches and molds. Gender differences were noted, with males more frequently sensitized to *Blomia tropicalis*, cockroaches, and molds, whereas females showed higher reactivity to *D. farinae* and animal dander. These findings highlight the influence of age and sex on allergen sensitization in this population. [71]
11. Study conducted by Liu et al, explored patterns of food and airborne allergen sensitization in 1,722 children aged 0–14 years in Sichuan province, Southwest China. Conducted between June and September 2019, it evaluated specific serum IgE responses to 10 food and 10 aeroallergens. Findings showed that nearly 60% of the children had sensitivity to at least one allergen, with food allergies (38.81%) being slightly more common than airborne allergies (24.90%). Notably, 36.28% were allergic to both types. Milk and egg

were the leading food allergens, while house dust mite and dust mite were the most frequent aeroallergens. Boys exhibited higher rates of inhalant allergies, and bronchitis emerged as the most common clinical manifestation. Sensitivity to food allergens peaked in children aged 0–2 years, while inhalant allergies were more prevalent in those aged 3–5 years. Nutritional status showed no significant difference between allergic and non-allergic children. These results highlight age and gender trends in allergen sensitization and underline the importance of early recognition for better management of childhood allergies. (72)

12. The study conducted by Gezmu AM et al conducted a retrospective cross-sectional study analyzed allergic diseases among children and adolescents treated at a single allergy center in Botswana over an eight-year period. Medical records of 407 patients aged 18 years or younger were reviewed. The majority were Black Africans (87.5%) with an average age of 5.8 years, and slightly more than half were female (58.7%). Asthma emerged as the most prevalent allergic condition (61.2%), followed by allergic rhinitis (57.0%) and atopic dermatitis (40.5%). Sensitization to common allergens—such as grass, molds, dust mites, and animal dander—was detected through skin prick tests, especially among asthmatic patients. Children with allergic rhinitis and conjunctivitis also reacted to tree pollen and the same allergens identified in asthmatics. Coexisting conditions were frequent, with asthma often accompanying allergic rhinitis, conjunctivitis, atopic dermatitis, and food allergies. Common asthma triggers included upper respiratory infections, weather changes, and secondhand smoke. A significant link was found between family history and the occurrence of allergic diseases—paternal allergy was associated with both asthma and rhinitis, while maternal history

correlated with atopic dermatitis. The findings highlight modifiable risk factors and provide a basis for future targeted interventions in pediatric allergy care. [73]

13. Study conducted by McColley et al was a prospective study aimed to explore the prevalence of allergic sensitization in children with habitual snoring and its potential link to obstructive sleep apnea syndrome (OSAS). Conducted at a pediatric pulmonary sleep clinic, the study included 39 children referred for overnight polysomnography. Each child underwent clinical evaluation, and serum samples were tested for allergic sensitization using a multiantigen RAST. Findings revealed that 36% of the participants had allergic sensitivities—higher than typical rates in the general pediatric population. Notably, children who tested positive for allergens had a higher incidence of OSAS (57%) compared to those without allergen sensitivity (40%), with the difference being statistically significant. The study concludes that allergy is commonly seen in children who snore regularly and may be a contributing risk factor for developing OSAS. [74]
14. Study conducted by Sporik R et al as a longitudinal study explored the relationship between early exposure to house-dust mite allergen (Der p I) and the later development of sensitization and asthma in children genetically predisposed to allergic diseases. Conducted in the UK from 1978 to 1989, the study followed a group of children with a family history of allergy. By 1989, among 67 children, 35 were found to be atopic, and 17 had active asthma—of whom 16 were sensitized to house-dust mites. All children with asthma, except one, had early-life exposure to dust mite allergen levels above 10 µg/g at age one, indicating a significant association between early allergen exposure and asthma risk (relative risk = 4.8). Higher exposure levels at age one were

also linked to earlier onset of wheezing. These findings highlight that, beyond genetic predisposition, early environmental exposure to house-dust mite allergens plays a key role in the development of asthma. [75]

15. Study conducted by Langley SJ et al examined the impact of in-home exposure to common indoor allergens (dust mites, cats, and dogs) on lung function and airway inflammation in individuals with asthma. A total of 311 participants underwent spirometry, bronchial reactivity tests, and exhaled nitric oxide (eNO) measurements. Dust samples from their homes were analyzed for allergen levels using ELISA. Findings revealed that asthmatic individuals who were both sensitized and exposed to high levels of the allergens had significantly reduced lung function, elevated eNO levels, and increased airway hyperresponsiveness compared to those who were not sensitized and exposed. Specifically, exposure to cat allergens in sensitized individuals significantly contributed to airway reactivity. The results indicate that living in environments with allergens one is sensitized to can exacerbate asthma severity. [76]
16. Study conducted by Sala KA et al as a retrospective case-control study analyzed children hospitalized for asthma over one year to identify factors linked to severe exacerbations requiring ICU care. Out of 188 hospitalized children, 57 (30%) were admitted to the ICU. While demographic factors like age, gender, and race were similar between ICU and ward patients, those in the ICU were more likely to have their attacks triggered by allergens or irritants. They also experienced a quicker onset of symptoms before hospitalization. These findings highlight that allergen-related triggers and rapid symptom development are key contributors to severe asthma episodes in children. [77]

17. Study conducted by Arshad SH et al explored the link between atopy—defined by a positive skin prick test response to allergens—and the development of asthma, rhinitis, and eczema in four-year-old children from a birth cohort of 1456 individuals in the UK. Of the 981 children tested with a panel of 12 common allergens, 28.1% had allergic conditions, and 19.6% were found to be atopic. The most common sensitizations were to house dust mite, grass pollen, and cat allergens. Sensitization to inhalant allergens was more prevalent than to food allergens. A strong correlation was observed between atopy and the presence of allergic diseases, particularly with exposure to house dust mite, which posed the highest risk for asthma. Sensitization to grass pollen was most associated with rhinitis, while peanut sensitization showed a strong link to eczema. Male children had a higher rate of atopy, especially for house dust mite and grass pollen. Additionally, the risk of developing allergic diseases increased with the number of allergens a child was sensitized to, suggesting a dose-response relationship. Interestingly, pet ownership did not significantly affect the rate of sensitization to cat or dog allergens. The study concludes that atopy, especially to potent allergens like house dust mite, plays a significant role in the early development of allergic diseases. [(78)]

18. Study conducted by Illi et al as a longitudinal study, part of the German Multicentre Allergy Study, tracked 1314 children from birth to age 13 to understand how early-life allergen sensitization and exposure influence long-term lung function in asthma. The findings revealed that children who experienced wheezing without allergic sensitization in early life typically outgrew their symptoms by school age and maintained normal lung function into adolescence. In contrast, those who became sensitized to year-round allergens—such as dust mites, cat or dog dander—within the first three years

were more likely to develop persistent asthma with reduced lung function by school age. The negative impact on lung function was particularly pronounced in children who were both sensitized and exposed to high levels of these allergens early in life. Key lung function indicators, including the FEV1/FVC ratio and MEF50, were significantly lower in this group compared to children without such sensitization or exposure. Moreover, these children were more likely to develop airway hyper-responsiveness. Importantly, allergen sensitization or exposure later in childhood and sensitization to seasonal allergens had a much smaller effect on long-term asthma outcomes.

The early years of life, especially the first three, are critical in shaping the trajectory of asthma. Persistent allergic airway inflammation during this period—driven by early sensitization and exposure to indoor allergens—contributes to the development of chronic asthma and long-term lung function decline. [(79)]

19. Study conducted by Mahesh PA investigated how environmental differences influence allergen sensitization among individuals of the same ethnic background residing in various regions of South India. A total of 546 patients with allergic rhinitis and/or asthma were evaluated using questionnaires, skin-prick testing for common airborne allergens, and spirometry. Participants were categorised based on whether they lived in urban, suburban, or rural settings. The most prevalent allergens identified were house dust mites (65–70%), tree pollen (52–56%), and cockroach allergens (39–53%). In individuals under 21 years old, those from suburban and rural areas had a significantly lower risk of cockroach sensitization compared to their urban counterparts. In contrast, the same age group from rural and suburban areas had a higher likelihood of sensitization to fungal allergens. While allergen sensitization patterns were

generally consistent across regions, notable differences were observed among younger individuals. Urban living was associated with increased cockroach sensitization, while rural and suburban environments were linked to higher fungal sensitization. These findings underscore the role of environmental exposure in shaping allergic sensitivity and highlight the need for further research into how gene-environment interactions contribute to allergic disease development. [7]

20. Study conducted by Roberts G et al used Skin prick testing (SPT), a key tool in identifying allergen sensitization, was used to track allergic development in children from the 1989 Isle of Wight birth cohort across ages 4, 10, and 18. The proportion of children sensitized to at least one allergen increased steadily over time—19.7% at age 4, 26.9% at age 10, and 41.3% by age 18—with boys showing a consistently higher rate. While some children outgrew their sensitivities, most showed a progressive pattern, often becoming sensitized to multiple allergens over time. Early sensitization, especially by age 4, strongly predicted future sensitization, suggesting that allergen sensitivity tends to persist and expand through adolescence. [80]
21. Study conducted by Sattar HA et al as a hospital-based prospective study in Qatar evaluated common indoor and outdoor allergens among 1,106 patients over the age of 12 with asthma and/or allergic rhinitis. Skin prick testing and IgE measurements revealed that 51.4% of participants had positive reactions to allergens. The most common clinical presentation was a combination of asthma and rhinitis (52%), followed by rhinitis alone (32%) and asthma alone (15%). House dust mites—Dermatophagoides pteronyssinus (41.6%) and Dermatophagoides farinae (36.9%)—were the most frequent allergens, followed by cockroach allergens (32.2%). Females had a higher prevalence of

asthma and skin conditions, while males showed a higher rate of allergic rhinitis and greater sensitization to mites and insects. Pollen, grasses, and molds were more common allergens in females. The study emphasizes the significant role of environmental allergens in respiratory conditions in Qatar and highlights the need to reduce exposure to manage disease severity effectively. [81]

22. Study conducted by Janahi et al aimed to assess the prevalence of asthma and allergic conditions among Qatari schoolchildren aged 6 to 14, using the ISAAC questionnaire. Conducted between February 2003 and February 2004, the cross-sectional study included 3,283 children from both urban and rural areas, with an average age of 9 years. Results showed high rates of asthma (19.8%), allergic rhinitis (30.5%), eczema (22.5%), and chest infections (11.9%). Boys had a higher prevalence of these conditions compared to girls, and asthma and allergic rhinitis tended to decrease with age. A similar pattern of allergic conditions was observed among the children's parents, with asthma being slightly more common in mothers than fathers. The asthma rate in Qatari children closely aligns with that of neighboring Oman but exceeds rates in several other developing nations. The findings suggest that genetic predispositions, such as high consanguinity, along with environmental and lifestyle factors, may contribute to the elevated rates of asthma and allergies in Qatar. [82]

23. Study conducted by Sharif HAA et al as a retrospective cross-sectional study aimed to identify the most frequent allergens causing sensitization in children through skin prick tests (SPTs). Data were collected from 180 pediatric patients diagnosed with allergic rhinitis (AR), asthma, or atopic dermatitis (AD) at University Hospital Sharjah between 2013 and 2015. SPTs were

conducted using 15 regionally relevant aeroallergens, and a wheal size of 3 mm or more than the negative control was considered positive. The majority of patients (69.4%) had AR, followed by asthma (52.2%) and AD (21.1%). Most of the sensitized children (89.4%) showed reactions to multiple allergens. House dust mites (HDMs) were the most common allergen, followed by cat dander and feather mix. Among indoor allergens, cockroach had the lowest sensitization rate (8.9%). For molds, *Alternaria alternata* had the highest rate (26.1%), while outdoor allergens like *Phleum pratense* (21.1%) and Russian thistle (17.2%) were also prevalent. Bermuda grass showed the lowest sensitization (5.6%) among outdoor triggers. The findings highlight HDMs as the leading cause of sensitization, with AR being the most common allergic condition observed. [83]

24. Study conducted by Al-Rabia MW aimed to determine the most common inhalant and food allergens among individuals with allergic symptoms in Jeddah, Saudi Arabia. A total of 209 patients referred to Alborg Laboratory were tested for specific IgE levels using the RIDA Allergy Screen, which measures allergy severity across six classes. Among these patients, 65.1% showed sensitivity to at least one allergen. House dust mites were the most prevalent indoor allergens, followed by American cockroach, cat dander, pollen (especially *Cynodon dactylon*), and mold (*Alternaria tenuis* being the most common). In terms of food allergens, peanut was the most frequently detected, followed by cocoa, egg white, cow milk, chocolate, wheat flour, pistachio, goat milk, and codfish. The findings highlight a high rate of sensitization to both aeroallergens and common food items in this population. [84]

25. Study conducted by Alkhater SA as a retrospective cross-sectional study analyzed the allergen sensitization patterns in 100 asthmatic children aged 5 to 14 years in Al-Khobar, Eastern Province of Saudi Arabia. Conducted between January 2011 and December 2012, skin prick tests revealed that house dust mites (54%), cat fur (53%), and German cockroaches (26%) were the most common indoor allergens. Among outdoor triggers, *Salsola kali* (48%) topped the list, followed by Timothy grass and *Chenopodium album* (23% each). *Prosopis glandulosa* was the most frequent tree allergen (19%), and *Alternaria* and *Aspergillus* species were the most prevalent molds (21% each). The findings indicate a high prevalence of aeroallergen sensitization in this population, likely influenced by environmental changes linked to rapid urbanization in the region. [(85)]

MATERIALS AND METHODS

Source of Data

Children with allergic respiratory airway diseases at a tertiary care centre, KAHER'S Dr. Prabhakar Kore hospital, Belgaum-590010, Karnataka were included in the study.

Definitions

Bronchial asthma

Children with symptoms of wheeze, shortness of breath, chest tightness or cough with confirmed FEV1/FVC <0.80, an increase in FEV1 by >200ml or >12%following bronchodilator therapy from the baseline confirmed by spirometry according to GINA guidelines 2022.

Allergic rhinitis

Children with symptoms of nasal congestion, itching, sneezing, clear rhinorrhoea and conjunctival irritation with no evidence of upper respiratory tract infection or structural abnormalities, occurring <4days/week and for < 4 consecutive weeks as intermittent and >4days/week and >4 consecutive weeks as persistent symptoms.

Study Design

Cross-sectional study

Study Period

1 year

Sample size

Sample size calculation was done using the formula:

N= (z^2 * pq)/ (d^2)

Where, z- standard normal variate value (2-1.96 at 5% alpha error),

d-margin of error (9%),

p-84.31%,

q=100-p = 15.69%

Hence, the sample size for the study was 63.

Inclusion criteria

- Children between 5-18 years of age who are a known case of isolated bronchial asthma
- Children between 5-18 years of age who are a known case of isolated allergic rhinitis
- Children between 5-18 years of age who are a known case of combined bronchial asthma and allergic rhinitis.

Exclusion criteria

- Children between 5-18 years of age with acute lower respiratory tract infection.
- Children between 5-18 years of age on oral steroid therapy and/ or immunosuppressants.
- Children between 5-18 years of age with obesity.

Study protocol

A one-year cross-sectional study in children with allergic respiratory disease at a tertiary care centre.

Data collection procedure

After obtaining institutional and ethical clearance and informed consent from the parents, a detailed history was obtained regarding the course of the disease, past history and previous exposure to allergens and a plain blood serum sample was obtained from the patient for the study. The samples were stored up to 14 days at +2 to +8 degree centigrade in the microbiology laboratory. Test kit used for performing the test was a rapid method of detecting IgE antibodies to specific inhalant allergens in the patient's serum. The test is a multi-parameter assay containing optimised combinations of relevant allergens that helps in simultaneous analysis of serum IgE against different allergens. The test kit used in this study contains test strips coated with different allergen extracts. The test strips are first moistened and then incubated with patient's samples in the first reaction step. If samples contain specific antibodies of class IgE, they will bind to the allergens coated on the strip. To detect the bound antibodies, a second incubation was carried out using an enzyme-labelled anti-human IgE catalysing a colour reaction which was then interpreted as the intensity of the bands and was calculated in EAST (Enzyme allergo sorbent test) classes of 0 to 6. The test results provided by a qualified microbiologist was interpreted for further assessment of the sensitization profile.

The classes divided based on the level of IgE detected:

Class	Concentration [kU/l]	Result
0	< 0.35	No specific antibodies detected.
1	0.35 ≤ sIgE < 0.7	Very low antibody titer, frequently no clinical symptoms where sensitisation is present.
2	0.7 ≤ sIgE < 3.5	Low antibody titer, existing sensitisation, frequently with clinical symptoms in the upper range of class.
3	3.5 ≤ sIgE < 17.5	Significant antibody titer, clinical symptoms usually present.
4	17.5 ≤ sIgE < 50.0	High antibody titer, almost always with clinical symptoms.
5	50.0 ≤ sIgE < 100.0	Very high antibody titer.
6	≥ 100.0	Very high antibody titer.

Data processing and analysis/ Statistical Analysis

The data collected was entered in the Excel. The continuous data was reported as mean and standard deviation. The categorical data was reported as frequency and percentage. Chi-square test was performed to test the association between two categorical variables. The p-value was considered as significant when it is less than 0.05.

RESULTS

Age and Gender

The mean age of the study participants was 8.71 ± 2.89 years. Majority of the study participants were in the age group of 5 to 11 years (n=50, 79.4%) followed by the age group 12 to 18 years (n=13, 20.6%).

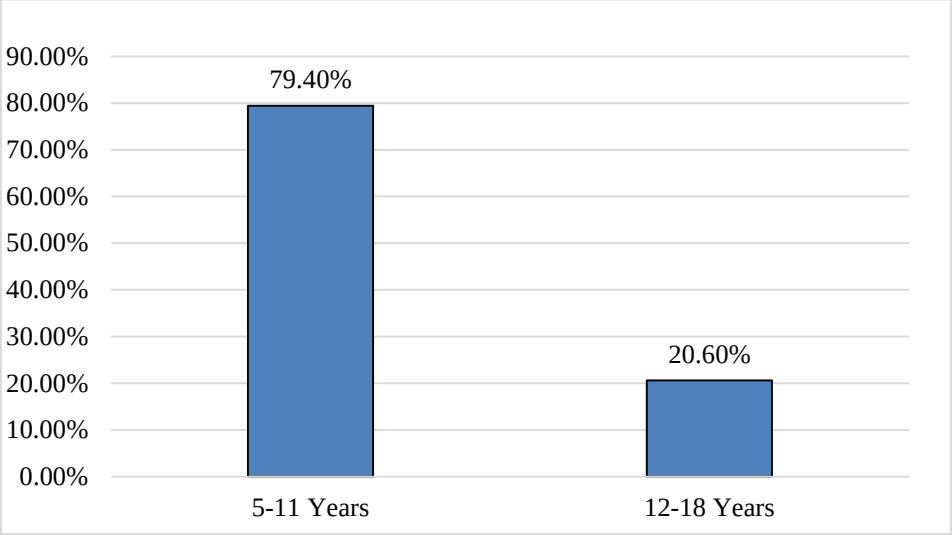

Figure 10: Distribution of age

Majority of the study participants were boys (n=41, 65.1%) (Girls: n=22, 34.9%).

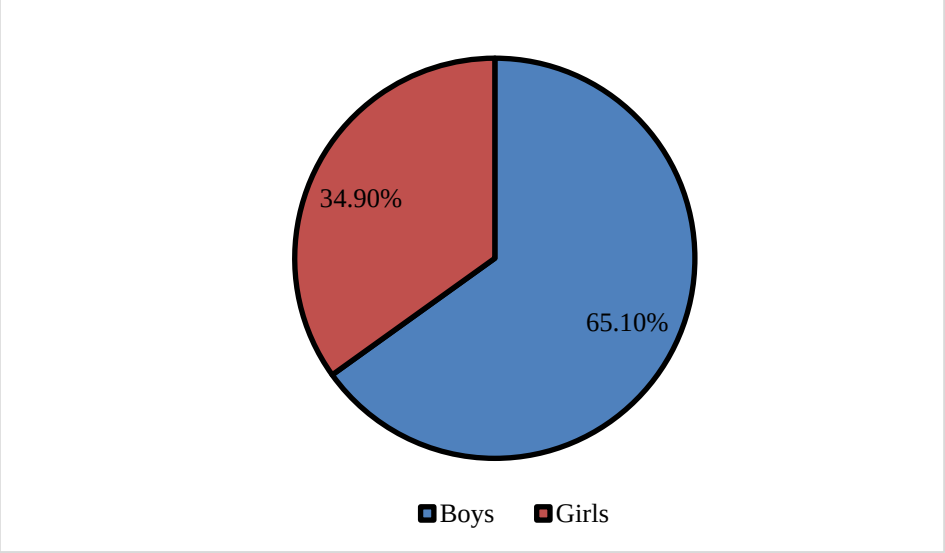

Figure 11: Distribution of gender

Geographical area

Among the study participants, 65.1% (n=41) were residing in the urban location and 34.9% (n=22) were residing in the rural location.

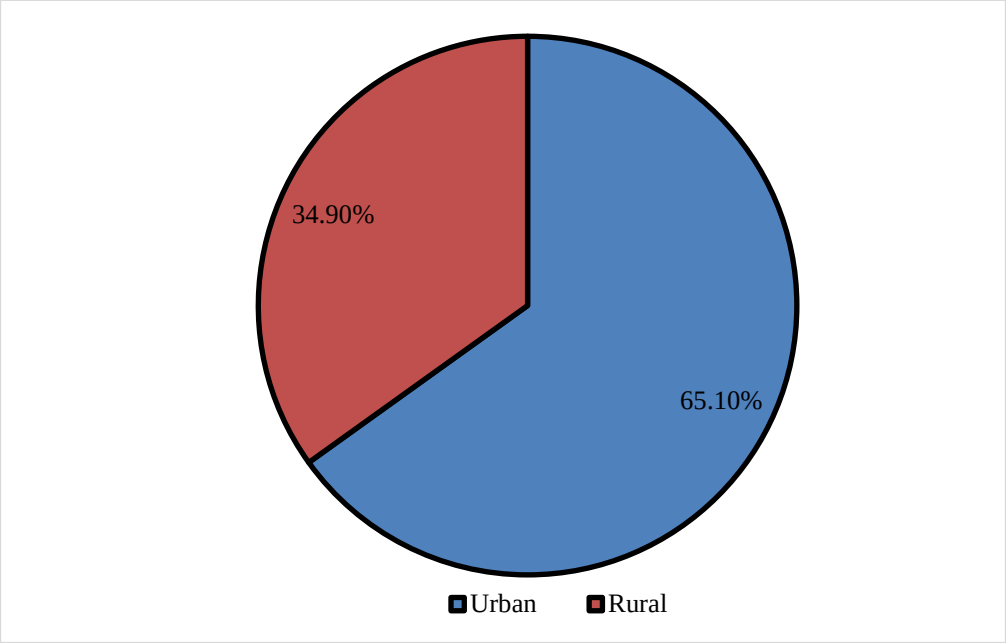

Figure 12: Geographic area of study participants

Allergens

The most common allergen was *Dermatophagoides pteronyssinus (Der pteronyssinus)* presenting in 27% (n=17) of the study participants, followed by 25.4% (n=16) *Dermatophagoides farinae (Der farinae)*, 9.5% (n=6) grass mix, 4.8% (n=3) mugwort, 4.8% (n=3) cat, 3.2% (n=2) horse, 3.2% (n=2) birch, 3.2% (n=2) *Alternaria alternata*, 1.6% (n=1) *Cladosporium herbarum*, and 1.6% (n=1) *Aspergillus fumigatus*.

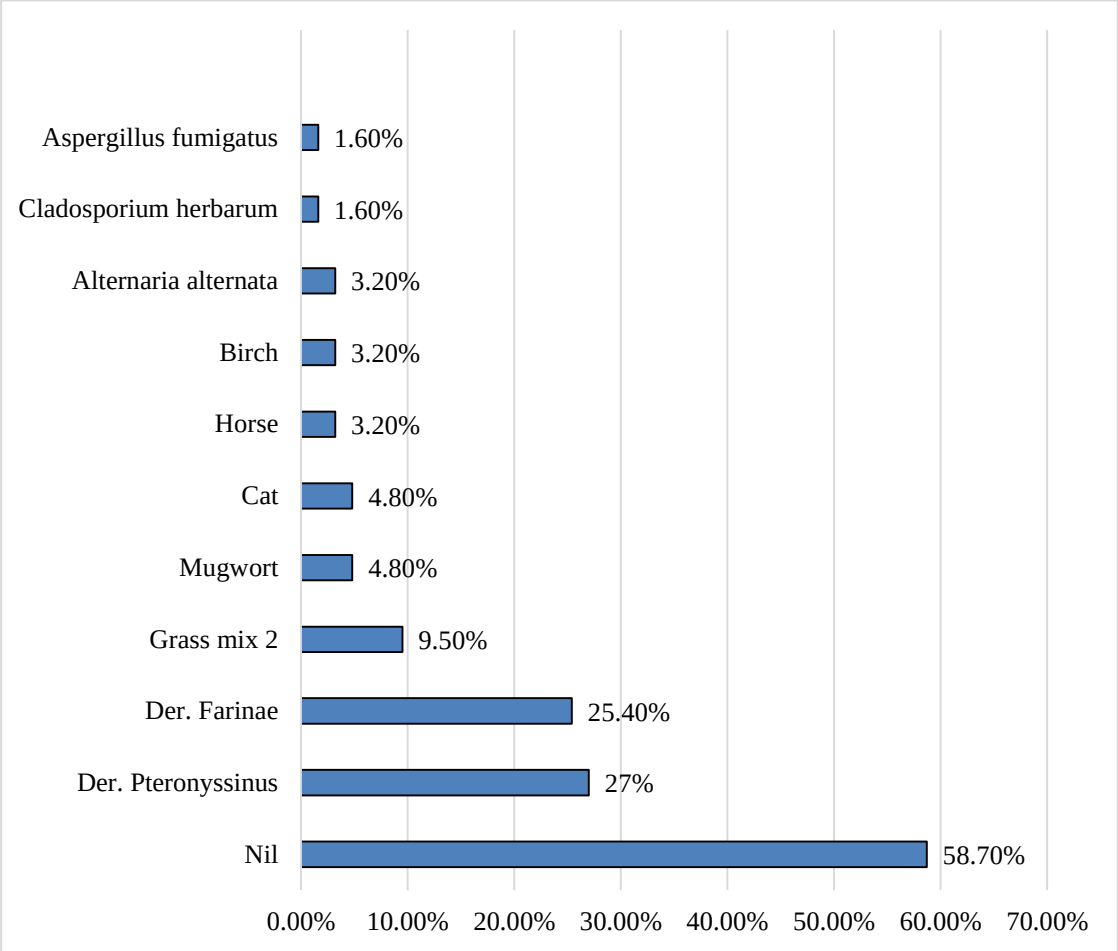

Figure 13: Allergen sensitivity

Table 4: Allergen sensitivity among study participants

Allergen	Class	Number (%)
Dermatophagoides pteronyssinus (n=17)	Class 1	4 (23.5%)
	Class 2	1 (5.9%)
	Class 3	3 (17.6%)
	Class 4	8 (47.1%)
	Class 5	1 (5.9%)
Dermatophagoides farinae (n=16)	Class 1	2 (12.5%)
	Class 2	4 (25%)
	Class 3	1 (6.3%)
	Class 4	5 (31.2%)
	Class 5	4 (25%)
Grass mix (n=6)	Class 1	4 (66.6%)
	Class 4	1 (16.7%)
	Class 5	1 (16.7%)
Mugwort (n=3)	Class 2	2 (66.7%)
	Class 3	1 (33.3%)
Cat (n=3)	Class 1	1 (33.3%)
	Class 2	1 (33.3%)
	Class 4	1 (33.3%)
Horse (n=2)	Class 1	1 (50%)
	Class 2	1 (50%)
Birch (n=2)	Class 1	2 (100%)
Alternaria alternata (n=2)	Class 1	2 (100%)
Cladosporium herbarum (n=1)	Class 1	1 (100%)
Aspergillus fumigatus (n=1)	Class 1	1 (100%)

Indoor and outdoor allergen

Among the allergens reported, 34 (64.12%) were indoor allergens and 19 (35.8%) were outdoor allergens. Among indoor allergens, *Dermatophagoides pteronyssinus* constitutes 50% (n=17), followed by *Dermatophagoides farinae* contributing 47.1% and *Aspergillus fumigatus* contributing 2.9%. Among outdoor allergens, 31.6% (n=6) was grass mix, followed by 15.8% (n=3) mugwort, 15.8% (n=3) cat, 10.5% (n=2) horse, 10.5% (n=2) birch, 10.5% (n=2) *Alternaria alternata* and 5.3% (n=1) *Cladosporium herbarum*.

Table 5: Indoor and outdoor allergens

Allergen	**Number (%)**
Indoor allergen	**34 (100%)**
1. *Dermatophagoides pteronyssinus*	17 (50%)
2. *Dermatophagoides farinae*	16 (47.1%)
3. Aspergillus fumigatus	1 (2.9%)
Outdoor allergen	**19 (100%)**
1. Grass mix	6 (31.6%)
2. Mugwort	3 (15.8%)
3. Cat	3 (15.8%)
4. Horse	2 (10.5%)
5. Birch	2 (10.5%)
6. Alternaria alternata	2 (10.5%)
7. Cladosporium herbarum	1 (5.3%)

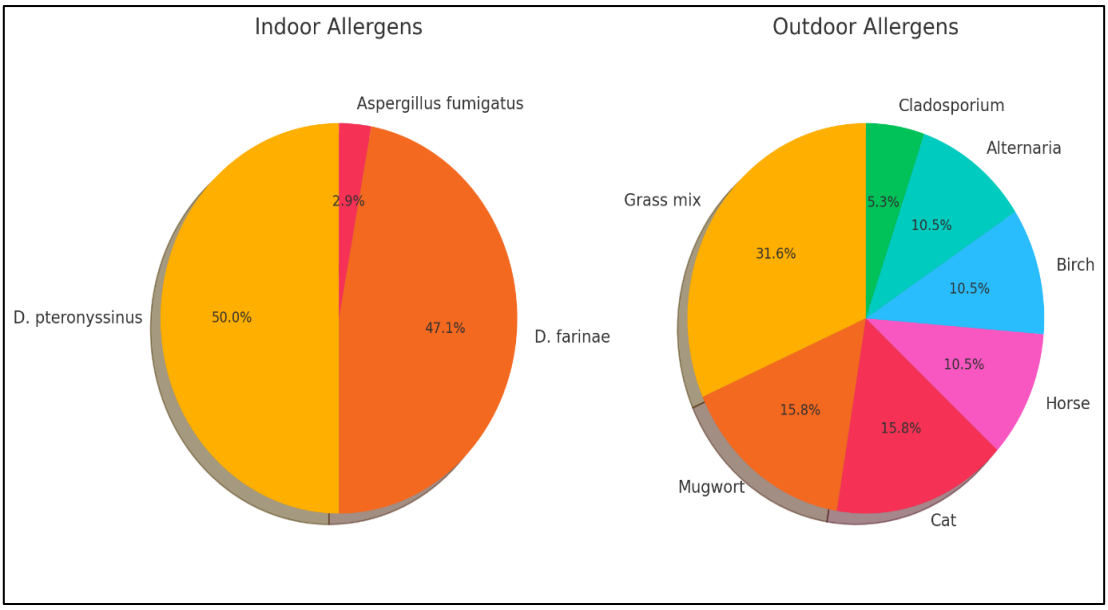

Figure 14: Indoor and outdoor allergens

Geographical area Vs Aeroallergen

Aeroallergen sensitization in urban study participants had shown 34.1% *Dermatophagoides pteronyssinus*, 31.7% *Dermatophagoides farinae*, 12.2% of grass mix, 7.3% of mugwort, 7.3% of cat, 4.9% of horse, 4.9% of birch, 2.4% of *Alternaria alternata* and 2.4% of *Aspergillus fumigatus*.

In the rural population, 13.6% of *Dermatophagoides pteronyssinus*, 13.6% of *Dermatophagoides farinae*, 4.5% of grass mix, 4.5% of *Alternaria alternata*, and 4.5% of *Cladosporium herbarum* was reported.

Table 6: Allergens and geographic area

Locality	**Urban (n=41)**	**Rural (n=22)**	**P Value**
No sensitivity (n=37)	20 (48.8%)	17 (77.3%)	**0.026**
Dermatophagoides pteronyssinus (n=17)	14 (34.1%)	3 (13.6%)	0.070
Dermatophagoides farinae (n=16)	13 (31.7%)	3 (13.6%)	0.140
Grass mix (n=6)	5 (12.2%)	1 (4.5%)	0.309
Mugwort (n=3)	3 (7.3%)	0 (0%)	0.268
Cat (n=3)	3 (7.3%)	0 (0%)	0.268
Horse (n=2)	2 (4.9%)	0 (0%)	0.420
Birch (n=2)	2 (4.9%)	0 (0%)	0.420
Alternaria alternata (n=2)	1 (2.4%)	1 (4.5%)	0.580
Cladosporium herbarum (n=1)	0 (0%)	1 (4.5%)	0.349
Aspergillus fumigatus (n=1)	1 (2.4%)	0 (0%)	0.651

Clinical presentation

Allergic rhinitis was diagnosed in 61.9% (n=39) of the study participants. Bronchial asthma was reported in 30.2% (n=19) of the study participants. United airway disease was present in 7.9% (n=5) of the study participants.

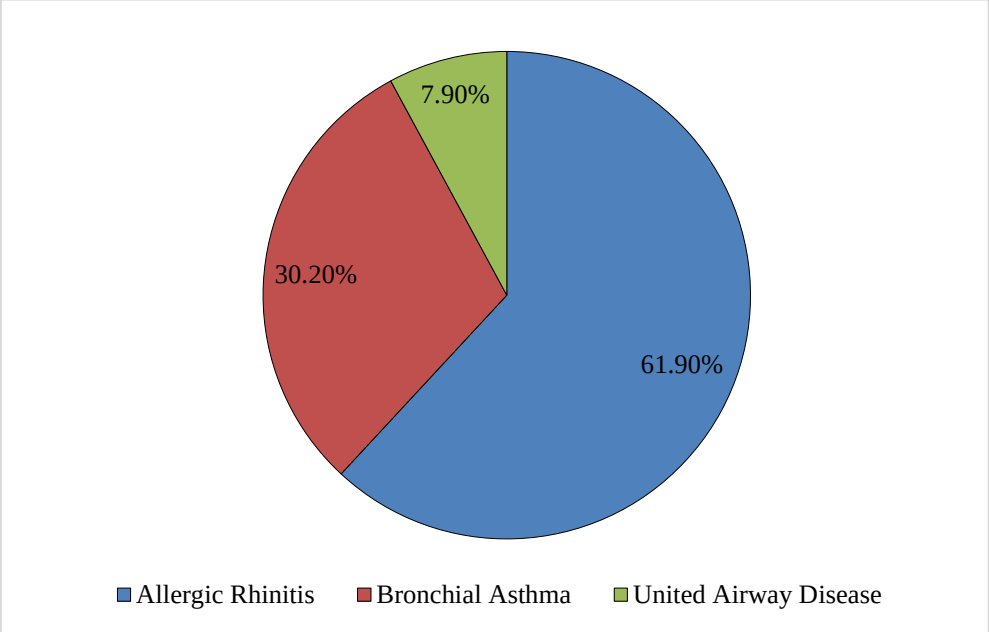

Figure 15: Clinical Presentation

Severity of disease

Allergic rhinitis

Severity of allergic rhinitis based on ARIA classification

Among the study participants with allergic rhinitis (n=39), majority of them were in the mild intermittent AR (n=29, 74.4%), followed by mild persistent AR (n=4, 10.2%), moderate to severe persistent AR (n=4, 10.2%) and moderate to severe intermittent AR (n=2, 5.2%).

Table 7: Severity of allergic rhinitis

Allergic rhinitis severity	Number (%)
Mild intermittent allergic rhinitis	29 (74.4%)
Mild persistent allergic rhinitis	4 (10.2%)
Moderate to severe intermittent allergic rhinitis	2 (5.2%)
Moderate to severe persistent allergic rhinitis	4 (10.2%)

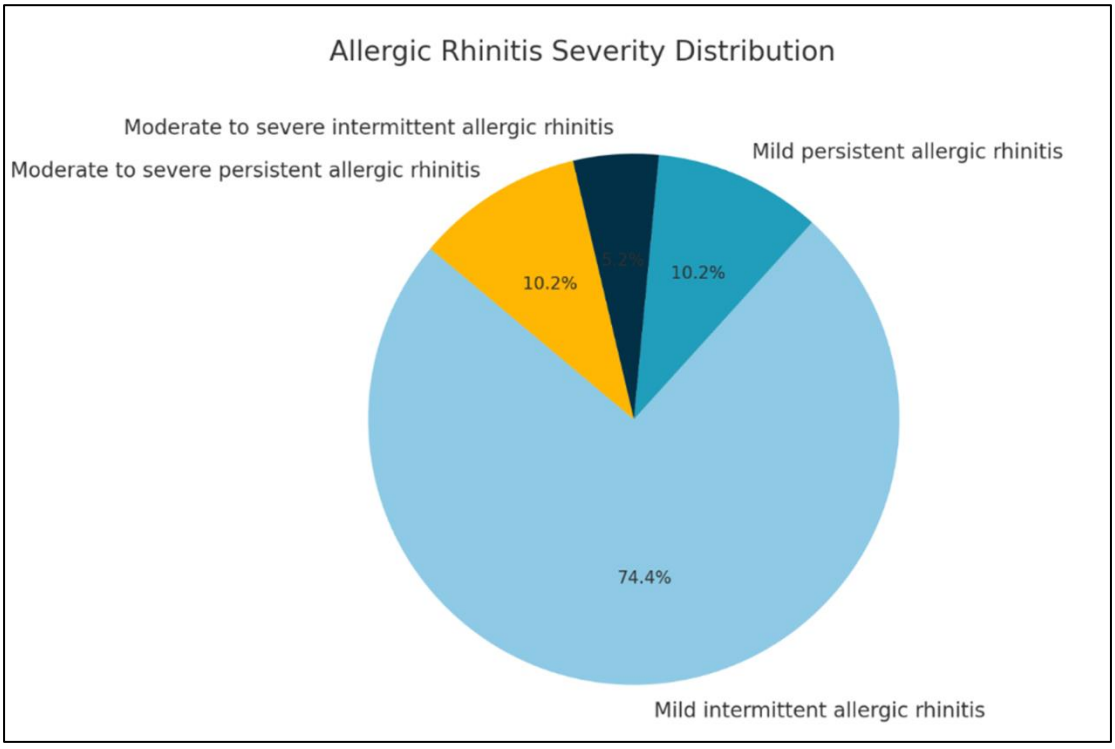

Figure 16: Severity of allergic rhinitis

Allergen vs allergic rhinitis

Among the study participants with allergic rhinitis, the most common allergens were *Dermatophagoides pteronyssinus* (n=8, 20.5%) and *Dermatophagoides farinae* (n=7, 17.9%).

Table 8: Allergens in allergic rhinitis

Allergens in allergic rhinitis	Number (%)
Dermatophagoides pteronyssinus	8 (20.5%)
Dermatophagoides farinae	7 (17.9%)
Grass mix	2 (5.1%)
Mugwort	1 (2.6%)
Cat	1 (2.6%)
Horse	2 (5.1%)
Birch	1 (2.6%)
Alternaria alternata	1 (2.6%)

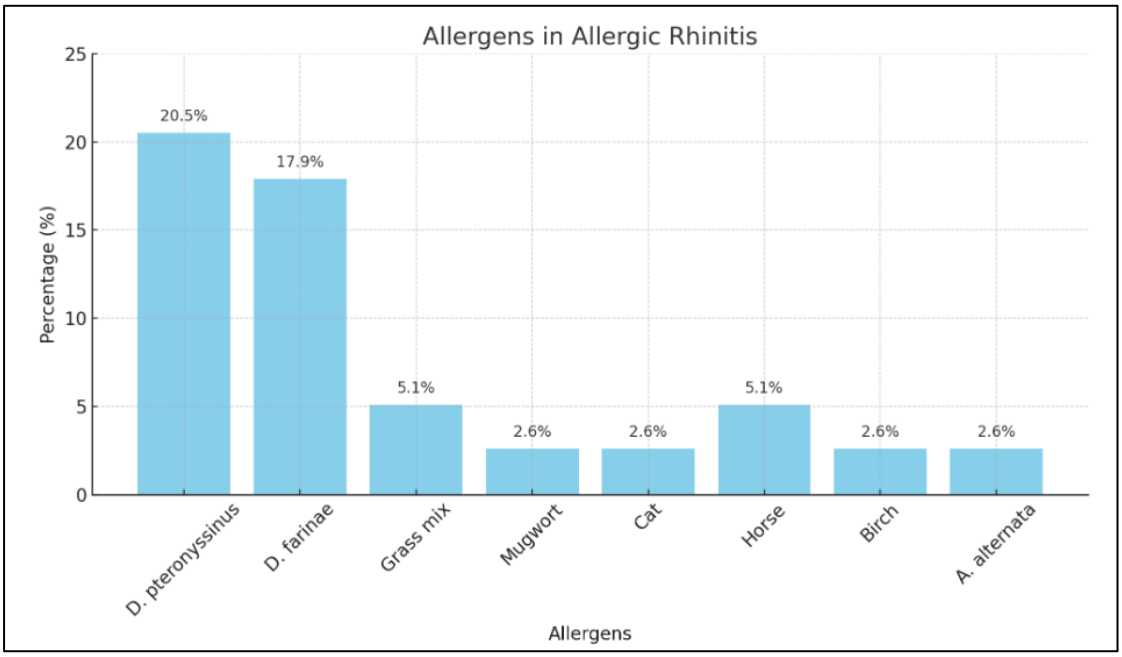

Figure 17: Allergens in allergic rhinitis

Most prevalent aeroallergens among severe AR study participants

Among the children with severe allergic rhinitis, the most common aeroallergens are identified as *Dermatophagoides pteronyssinus* (n=5, 83.3%) and *Dermatophagoides farinae* (n=5, 83.3%) being responsible for the severe disease outcome.

Table 9: Common aeroallergens among severe AR

Aeroallergens among severe AR n (6)	**Number (%)**
Dermatophagoides pteronyssinus	5 (83.3%)
Dermatophagoides farinae	5 (83.3%)
Horse	2 (33.3%)
Grass mix 2	1 (16.7%)
Birch	1 (16.7%)
Mugwort	1 (16.7%)

*Multiple allergens reported per individual

Allergic rhinitis severity vs Class

Among the study participants with mild intermittent AR, 10.3% and 3.4% constitutes Class 1 and Class 2 respectively. Among mild persistent AR, 50%, 16.7% and 16.7% were study participants in Class 1, Class 2 and Class 3 respectively. In moderate to severe intermittent AR, 33.3% each in Class 1, Class 2 and Class 3 respectively. In Moderate to severe persistent AR, 25%, 16.7%, 16.7%, 25% and 16.7% were Class 1, Class 2, Class 3, Class 4 and Class 5 respectively. The AR severity was significantly associated with the Class of allergen (p=0.000).

Table 10: Allergic rhinitis severity and class

Class	**AR severity**				**P Value**
	Mild intermittent AR	**Mild persistent AR**	**Moderate to severe intermittent AR**	**Moderate to severe persistent AR**	
Nil	25 (86.2%)	1 (16.7%)	0 (0%)	0 (0%)	**0.000**
Class 1	3 (10.3%)	3 (50%)	1 (33.3%)	3 (25%)	
Class 2	1 (3.4%)	1 (16.7%)	1 (33.3%)	2 (16.7%)	
Class 3	0 (0%)	1 (16.7%)	1 (33.3%)	2 (16.7%)	
Class 4	0 (0%)	0 (0%)	0 (%)	3 (25%)	
Class 5	0 (0%)	0 (0%)	0 (0%)	2 (16.7%)	

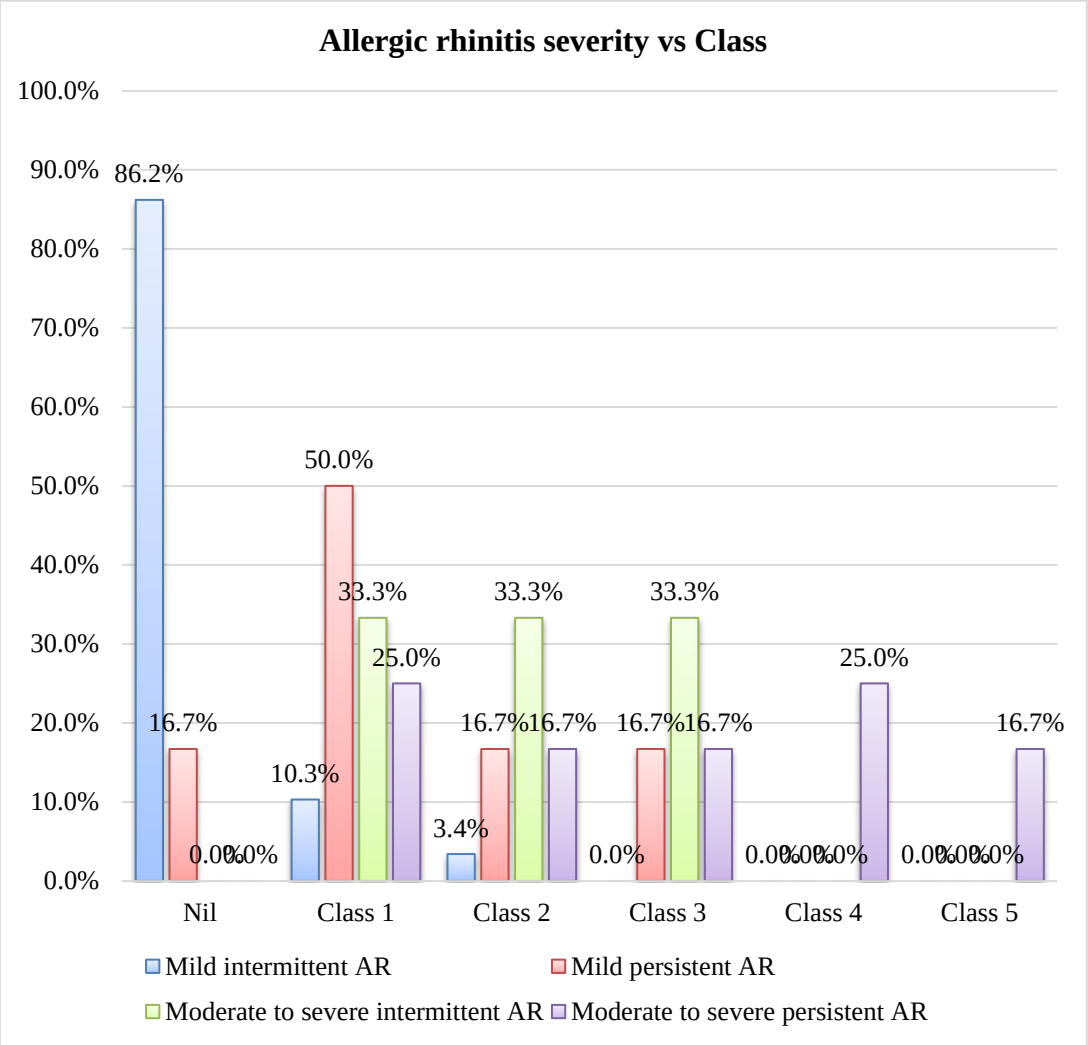

Figure 18: Class and severity of AR

Allergic rhinitis vs exacerbation

In mild persistent AR, 25% of the study participants had 1 episode of exacerbation in the past one year. In moderate to severe intermittent AR, 100% of the study participants had 1 episode of exacerbation in the past year. In moderate to severe persistent AR, 75% of the study participants had 2 episodes of exacerbations and 25% of the study participants had 3 episodes of exacerbations in the past one year. The number of exacerbations in the past year is significantly associated with severity of the disease (p=0.000).

Table 11: Exacerbations and severity of AR

Exacerbation in past 1 year	AR severity				P Value
	Mild intermittent AR	Mild persistent AR	Moderate to severe intermittent AR	Moderate to severe persistent AR	
0	29 (100%)	3 (75%)	0 (0%)	0 (0%)	0.000
1	0 (0%)	1 (25%)	2 (100%)	0 (0%)	
2	0 (0%)	0 (0%)	0 (0%)	3 (75%)	
3	0 (0%)	0 (0%)	0 (0%)	1 (25%)	

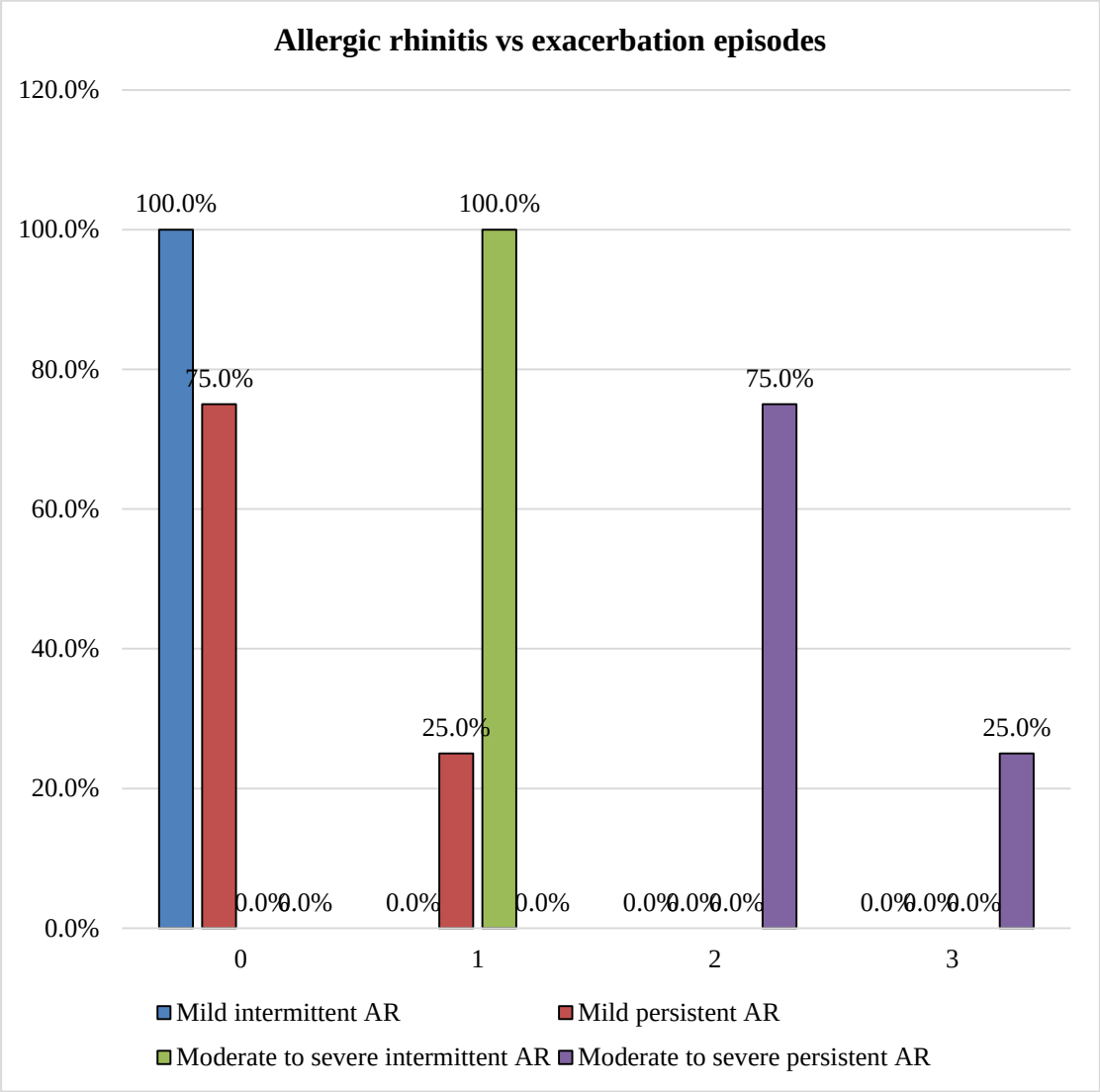

Figure 19: Allergic rhinitis and exacerbation episodes

Bronchial asthma

Severity of disease

Prevalence of mild, moderate and severe bronchial asthma was 63.2% (n=12), 15.8% (n=3) and 21% (n=4) respectively.

Table 12: Severity of BA

Bronchial asthma severity	Number (%)
Mild	12 (63.2%)
Moderate	3 (15.8%)
Severe	4 (21%)

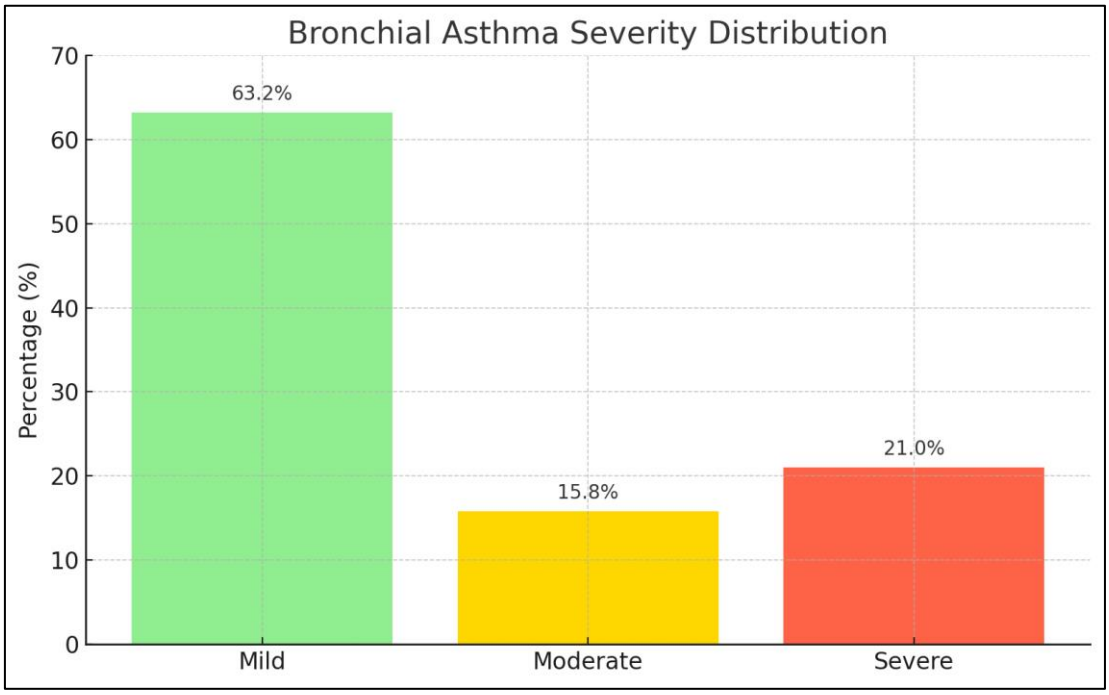

Figure 20: Bronchial asthma severity

Allergen vs bronchial asthma

The most common allergen among the study participants with bronchial asthma were *Dermatophagoides pteronyssinus* (n=6, 31.6%) and *Dermatophagoides farinae* (n=6, 31.6%).

Table 13: Allergens in BA

VARIABLES	BRONCHIAL ASTHMA (n=19)
Dermatophagoides pteronyssinus	6 (31.6%)
Dermatophagoides farinae	6 (31.6%)
Grass mix	4 (21.1%)
Mugwort	1 (5.3%)
Cat	2 (10.5%)
Birch	1 (5.3%)
Alternaria alternata	1 (5.3%)
Cladosporium herbarum	1 (5.3%)
Aspergillus fumigatus	1 (5.3%)

Severity vs class

Among mild bronchial asthma study participants, 26.7% and 20% were class 1 and Class 2 of allergens. Among moderate bronchial asthma study participants, 40%, 20% and 20% were Class 2, Class 4 and Class 5 respectively. Among severe bronchial asthma study participants, 28.6%, 14.3%, 50% and 7.1% were of Class 1, Class 2, Class 4 and Class 5 respectively. The severity of the disease was significantly associated with the class of the allergen (p=0.008).

Table 14: Severity of BA and Class

Class	BA severity			P Value
	Mild BA	Moderate BA	Severe BA	
Nil	8 (53.3%)	1 (20%)	0 (0%)	**0.008**
Class 1	4 (26.7%)	0 (0%)	4 (28.6%)	
Class 2	3 (20%)	2 (40%)	2 (14.3%)	
Class 4	0 (0%)	1 (20%)	7 (50%)	
Class 5	0 (0%)	1 (20%)	1 (7.1%)	

Most prevalent aeroallergens among severe BA study participants

Among the severe bronchial asthma study participants, the most common aeroallergens are *Dermatophagoides pteronyssinus* (n=4, 100%) and *Dermatophagoides farinae* (n=4, 100%), resulting in a severe disease outcome.

Table 15: Common aeroallergens in severe BA

Common aeroallergen (n=4)	**Number (%)**
Dermatophagoides pteronyssinus	4 (100%)
Dermatophagoides farinae	4 (100%)
Cat	2 (50%)
Grass mix 2	1 (25%)
Birch	1 (25%)
Mugwort	1 (25%)
Aspergillus fumigatus	1 (25%)

*Multiple allergens reported per study participants

Bronchial asthma vs exacerbation

One episode of exacerbation was reported in the 8.3% of mild bronchial asthma study participants in the past year. In mild bronchial asthma study group, one and two episodes of exacerbation was reported in 33.3% each. In severe bronchial asthma group, 50%, 25% and 25% of study participants had 1 episode, 2 episode and 3 episodes of exacerbation in the past year. The number of exacerbations were statistically significant with the severity of disease (p=0.024).

Table 16: BA and exacerbations

Exacerbation in one year	**BA severity**			**P Value**
	Mild BA	**Moderate BA**	**Severe BA**	
Nil	11 (91.7%)	1 (33.3%)	0 (0%)	**0.024**
1	1 (8.3%)	1 (33.3%)	2 (50%)	
2	0 (0%)	1 (33.3%)	1 (25%)	
3	0 (0%)	0 (0%)	1 (25%)	

United airway disease

Severity

Among the study participants with united airway disease (n=5), mild BA+ mild persistent AR was present in 20% (n=1), Moderate BA + Mild intermittent AR was present in 20% (n=1), Moderate BA + Mild persistent AR was present in 20% (n=1), Moderate BA + Moderate to severe persistent AR was present in 20% (n=1), and Severe BA + Mild persistent AR was present in 20% (n=1) of the study participants with UAD.

Table 17: Severity of UAD

Severity of UAD	Number (%)
Mild BA + Mild persistent AR	1 (20%)
Moderate BA + Mild intermittent AR	1 (20%)
Moderate BA + Mild persistent AR	1 (20%)
Moderate BA + Moderate to severe persistent AR	1 (20%)
Severe BA + Mild persistent AR	1 (20%)

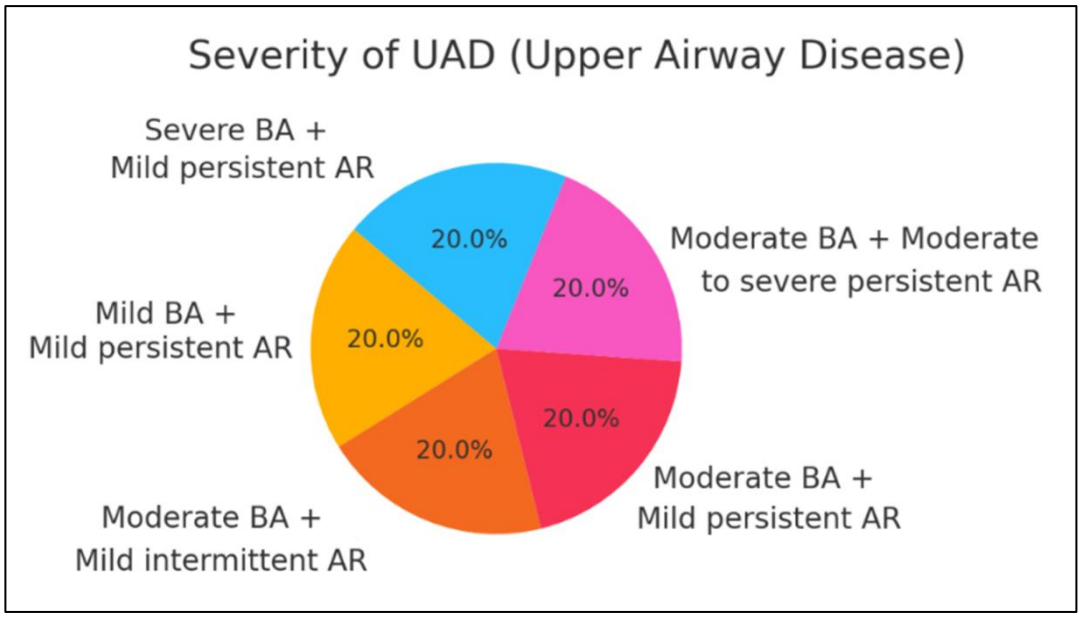

Figure 21: Severity of UAD

United airway disease vs allergens

The most common allergens in united airway disease were *Dermatophagoides pteronyssinus* (n=3, 60%) and *Dermatophagoides farinae* (n=3, 60%), identified to be the associated with severe disease outcome.

Table 18: Allergens in UAD

VARIABLES	UNITED AIRWAY DISEASE (n=5)
Dermatophagoides pteronyssinus	3 (60%)
Dermatophagoides farinae	3 (60%)
Mugwort	1 (20%)

Most prevalent aeroallergens in severe United airway disease

Among the children with severe united airway disease, the most common aeroallergens were *Dermatophagoides pteronyssinus* (n=2, 100%) and *Dermatophagoides farinae* (n=2, 100%) resulting in severe disease outcome.

Aeroallergens in severe UAD	**Number (%)**
Dermatophagoides pteronyssinus	2 (100%)
Dermatophagoides farinae	2 (100%)
Mugwort	1 (50%)

*Multiple aeroallergens reported per individual

United airway disease vs class

The moderate BA + mild persistent AR had 50% study participants in Class 4. Moderate BA + Moderate to severe persistent AR had 100% study participants in Class 3 and 50% study participants in class 4. Severe BA + Mild persistent AR had 100% study participants in Class 5.

Table 19: UAD and Class

United airway disease	Class				P value
	Nil	Class 3	Class 4	Class 5	
Mild BA + Mild persistent AR	1 (50%)	0 (0%)	0 (0%)	0 (0%)	0.077
Moderate BA + Mild intermittent AR	1 (50%)	0 (0%)	0 (0%)	0 (0%)	
Moderate BA + Mild persistent AR	0 (0%)	0 (0%)	2 (50%)	0 (0%)	
Moderate BA + Moderate to severe persistent AR	0 (0%)	1 (100%)	2 (50%)	0 (0%)	
Severe BA + Mild persistent AR	0 (0%)	0 (0%)	0 (0%)	2 (100%)	

Sensitization of study participants

Among the study participants, 12.7% (n=8) had one allergen and 28.6% (n=18) had two or more allergen. In 58.7% (n=37) of the study participants, no sensitization was found.

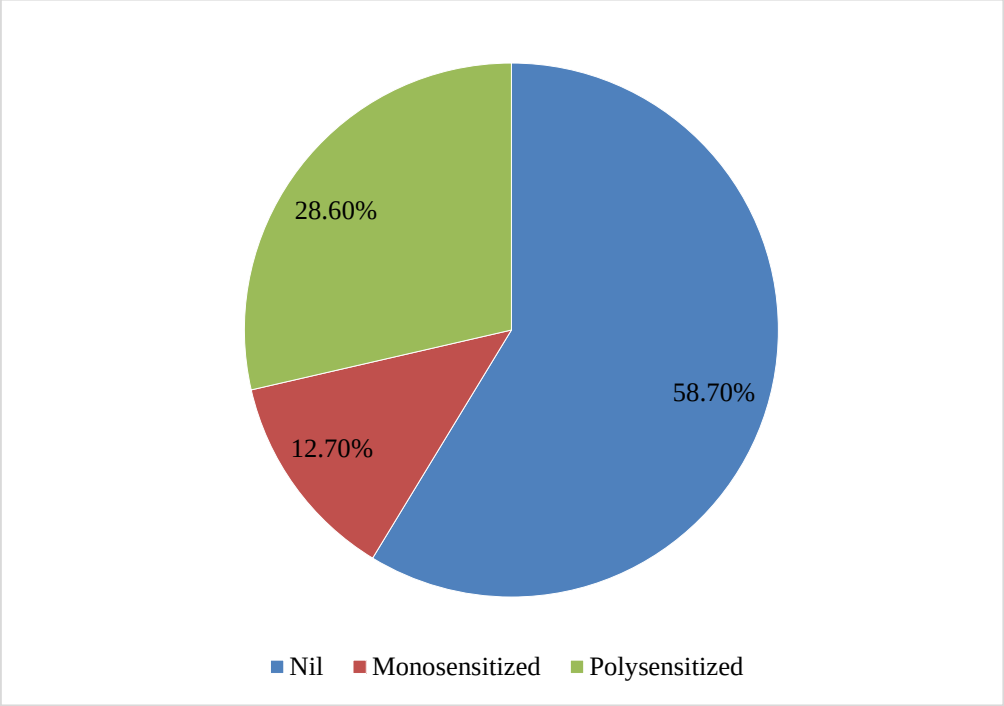

Figure 22: Sensitization

Clinical presentation and number of sensitizations

In monosensitization group the prevalence of allergic rhinitis, and bronchial asthma were 75% (n=6), and 25% (n=2) respectively. In polysensitization group, prevalence of allergic rhinitis, bronchial asthma and united airway disease were 38.9% (n=7), 44.4% (n=8) and 16.7% (n=3) respectively.

Table 20: Clinical presentation and number of sensitizations

DISEASE	SENSITIZATION			P VALUE
	NO	MONO	POLY	
Allergic rhinitis	26 (70.3%)	6 (75%)	7 (38.9%)	0.162
Bronchial asthma	9 (24.3%)	2 (25%)	8 (44.4%)	
United airway disease	2 (5.4%)	0 (0%)	3 (16.7%)	

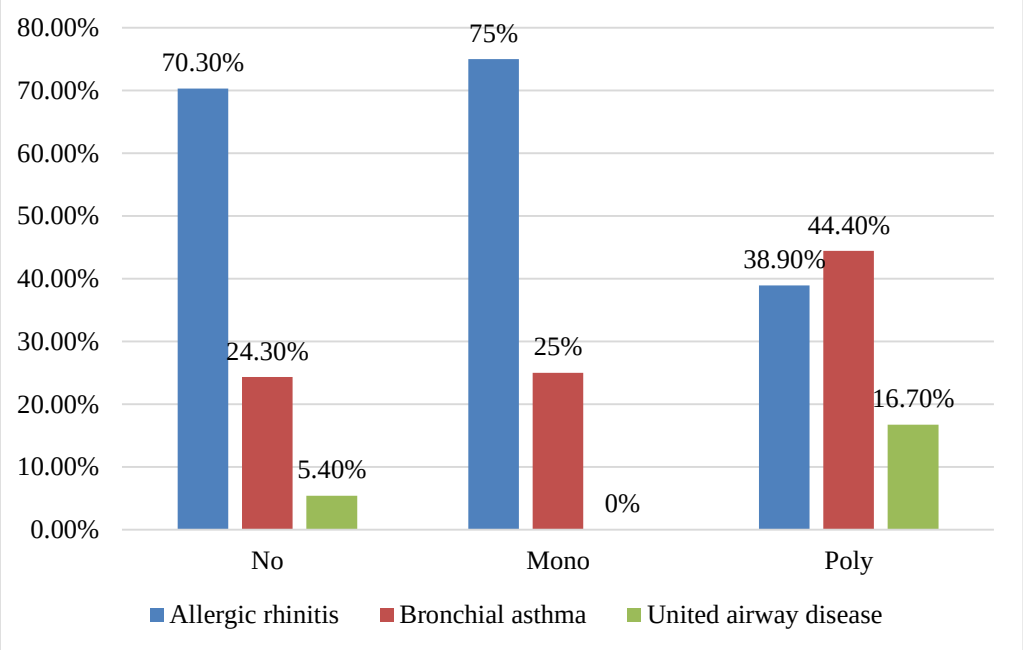

Figure 23: Clinical presentation and number of sensitizations

Clinical presentation compared with age and gender

The clinical presentations of allergic rhinitis, bronchial asthma and united airway disease were compared with age and gender. The results are tabulated as Table below.

Table 21: Clinical presentation compared with age and gender

VARIABLES	ALLERGIC RHINITIS (n=39)	BRONCHIAL ASTHMA (n=19)	UNITED AIRWAY DISEASE (n=5)	P value
Age				
5-11 Years	34 (87.2%)	12 (63.2%)	4 (80%)	0.105
12-18 Years	5 (12.8%)	7 (36.8%)	1 (20%)	
Gender				
Male	24 (61.5%)	13 (68.4%)	4 (80%)	0.671
Female	15 (38.5%)	6 (31.6%)	1 (20%)	

Class severity and level of treatment

The class range and treatment level when compared, were statistically significant (p=0.046).

Table 22: Class and treatment level

CLASS	TREATMENT LEVEL			P VALUE
	Mild	Moderate	Severe	
Nil	30 (81.1%)	4 (10.8%)	3 (8.1%)	**0.046**
Class 1-4 (Moderate)	13 (61.9%)	3 (14.3%)	0 (0%)	
Class 5 (Severe)	2 (40%)	0 (0%)	3 (60%)	

Disease severity and Treatment level

The severity of disease was compared with the level of treatment. It was statistically significant with p value of 0.000.

Table 23: Severity of disease and treatment level

DISEASE SEVERITY	TREATMENT LEVEL			P VALUE
	Mild	Moderate	Severe	
Mild	39 (84.8%)	7 (15.2%)	0 (0%)	**0.000**
Moderate	0 (0%)	0 (0%)	4 (100%)	
Severe	6 (46.2%)	0 (0%)	7 (53.7%)	

Exacerbation and Disease severity

Among the study participants, more than one episode of exacerbation was reported in 4.3%, 25% and 53.8% of mild, moderate and severe disease groups respectively. The disease severity was found to be statistically significant and was associated with one or more episodes of exacerbation.

Table 24: Exacerbations and disease severity

EXACERBATION	DISEASE SEVERITY			P VALUE
	Mild	Moderate	Severe	
None	41 (89.1%)	2 (50%)	3 (23.1%)	**0.000**
One episode	3 (6.5%)	1 (25%)	3 (23.1%)	
More than 1 episode	2 (4.3%)	1 (25%)	7 (53.8%)	

Exacerbation and Class

Among the study participants, more than one episode of exacerbation was reported in Class 1-4 in 23.8% (n=5) and 100% in Class 5 (n=5). The higher the class, the higher the episodes of exacerbations (p=0.000).

Table 25: Exacerbations and Class level

EXACERBATION	CLASS LEVEL			P VALUE
	Nil	Class 1-4	Class 5	
None	37 (100%)	9 (42.9%)	0 (0%)	**0.000**
One episode	0 (0%)	7 (33.3%)	0 (0%)	
More than 1 episode	0 (0%)	5 (23.8%)	5 (100%)	

DISCUSSION

Age and Gender

The mean age of study participants in the present study was 8.71 ± 2.89 years. Majority of the study participants were in the age group of 5 to 11 years (n=50, 79.4%) followed by the age group 12 to 18 years (n=13, 20.6%). Similar mean age was reported in the study conducted by Feliu A et al study was 8.4 ± 2.96 years (64) and a median age of the study participants in Dai L et al study was 8.8 (7.4-10.6) years (65). Lower mean age was reported in the study participants of Zahraldin et al with 7.6 ± 3.3 years.[86] Higher mean ages were reported in few studies. The mean age of the study participants in Bhagyashree P et al study was 11.26 ± 3.28 years.[66] The mean age of the study participants in Yadav S et al study was 9.28 ± 2.94 years.[87] The mean age of the study participants in allergic rhinitis, bronchial asthma and both allergic rhinitis & bronchial asthma in the study conducted by Umanets TR et al were 9.43 ± 0.55, 9.23 ± 3.63 and 8.55 ± 0.91 years respectively.[63]

In the present study, majority of the study participants were boys (n=41, 65.1%). Similarly, majority of the study participants were boys in Umanets TR et al., (n=57, 61.3%)[63], Bhagyashree P et al (n=63, 74%)[66], Dai L et al (n=109, 59.9%) [65], Zahraldin et al (n=327, 69.1%)[86], and Yadav S et al (n=46, 76.7%).[87] One study on the contrary had higher prevalence of female (girls=120, 64.2%).[64]

In the present study, among the study participants, 65.1% (n=41) were residing in the urban location and 34.9% (n=22) were residing in the rural location. In the study conducted by Meher BK et al 2021 majority of the study participants were from rural (67.5%).[88] In Raj D et al study, 23.8%, 72% and 3.2% of the study participants were from urban, urban slum and rural population.[10]

Clinical symptoms

In the present study, cold was the common clinical symptom reported 68.3% (n=43). Similarly, Kumar D et al study reported cold as common symptom (56.8%), followed by sneezing/ rhinorrhoea/ nasal obstruction in 48.9% and breathlessness in 30.3%. [(67)]

Precipitating factors

The common precipitating factor among the study participants was house dust presenting in 44.4% (n=28) in the present study. In the study conducted by Bhagyashree P et al., the upper respiratory tract infection (n=80, 94%) was the most common precipitating factor.[(66)] House dust mite was the most common allergen in all age groups in Zahraldin et al.[(86)]

Clinical presentation

Family history of asthma is an important factor in pathogenesis of the disease. However, the current study did not record the family history of the disease. However, few studies conducted by Zahraldin et al [(86),] Bhagyashree P et al [(66),] and Dai L et al. [(65)] had reported and reported positive correlation between them. Prevalence of allergic rhinitis, bronchial asthma and allergic rhinitis & bronchial asthma in the present study was 61.9%, 30.2%, and 7.9% respectively. Prevalence of allergic rhinitis, bronchial asthma and allergic rhinitis & bronchial asthma in the study conducted by Umanets TR et al., was 54.8%, 33.4%, and 11.8%.[(63)] Prevalence of allergic rhinitis, bronchial asthma and allergic rhinitis & bronchial asthma in the study conducted by Feliu A et al., was 21.9%, 10.2% and 65.8% respectively.[(64)] Allergic rhinitis was present in 34% (n=29) of the study participants in Bhagyashree P et al

study.[66] Prevalence of asthma, allergic rhinitis and both were 37%, 12% and 44.2% respectively in the Zahraldin et al study.[86] In Uysal P et al study, the prevalence of asthma, allergic rhinitis and both asthma & allergic rhinitis were 16.9%, 29.4% and 42.9% respectively.[89] In Kumar D et al study, 60.3% of the study participants had respiratory system involvement in any one of the following forms: allergic rhinitis, asthma or allergic rhinitis & asthma.[67]

In the present study, the prevalence of mild, moderate and severe bronchial asthma was 63.2% (n=12), 15.8% (n=3) and 21% (n=4) respectively. In the study conducted by Raj D et al, the prevalence of intermittent, mild persistent, moderate persistent and severe persistent asthma was 2.2%, 33.9%, 53.3%, and 10.5% respectively.

Sensitization of study participants

Among the study participants in the present study, 12.7% (n=8) had one allergen and 28.6% (n=18) had two or more allergen. In 58.7% (n=37) of the study participants, no sensitization was found in the present study. In Umanets TR et al., study, 82.8% (n=77) had more than one group of allergens.[63] In Feliu A et al., study, 57.8% of the study participants are polysensitized and 30% of the study participants were sensitized to both food and pollens.[64] In Bhagyashree P et al., study, 74% (n=63) of the study participants tested positive for one or more antigens in the skin prick test.[66] In Dai L et al study, 67.4% (n=155) of the study participants had at least one positive skin response and among the positive skin response, 7.7% had one allergen, 14.2% had two allergens, 19.4% had three allergens, 20% had four allergens and 38.7% had five or more than five allergens.[65] In the study conducted by Zahraldin et al., 43.1% were monosensitized, 45.5% were oligosensitized and 11.4%

were polysensitized.[86] In Yadav S et al study, 60% of the skin prick test was positive with one allergen in 40%, two allergens in 3.33%, three allergens in 30%, and four allergens in 26.66%.[87] In Uysal P et al study, 38.3% of the study participants had monosensitization and the rest 61.7% had polysensitization.[89] In Raj D et al study, 55.6% of the study participants were positive to at least one allergen and 37.8% of the study participants were positive to more than one allergen.[10]

Allergens

The most common allergen was in the present study was *Dermatophagoides pteronyssinus* in 27% (n=17) followed by 25.4% (n=16) *Dermatophagoides farinae.* Similarly, *Dermatophagoides pteronyssinus* was the common allergen in studies conducted by Bhagyashree P et al., (44%),[66] Dai L et al (59.3%),[65] Zahraldin et al (38.41%),[86] Yadav S et al.[87] In Uysal P et al study, among the fungal allergen sensitization, *Alternaria alterna* (45.5%) was the commonest and in non-fungal allergen sensitization, grass (82.3%) was the most common allergen.[89] Housefly was the common allergen found in the study conducted by Raj D et al.[10] House dust mites were the common allergen among respiratory system in the study conducted by Kumar D et al (54.7%).[67]

In our study, the number of allergens the individual is sensitized to was not significant with the clinical presentation. However, a study conducted by Raj D et al showed the number of sensitizations was significant with age.[10]

In the present study with allergic rhinitis, the most common allergens were *Dermatophagoides pteronyssinus* (n=8, 20.5%) and *Dermatophagoides farinae* (n=7, 17.9%).

In the present study the most common allergen among the study participants with bronchial asthma were *Dermatophagoides pteronyssinus* (n=6, 31.6%) and *Dermatophagoides farinae* (n=6, 31.6%).

In the present study the most common allergens in united airway disease were *Dermatophagoides pteronyssinus* (n=3, 60%) and *Dermatophagoides farinae* (n=3, 60%).

Indoor and outdoor allergen

Among the allergens reported, 34 (64.12%) were indoor allergens and 19 (35.8%) were outdoor allergens. Among indoor allergens, *Dermatophagoides pteronyssinus* constitutes 50% (n=17), followed by *Dermatophagoides farinae* contributing 47.1% and *Aspergillus fumigatus* contributing 2.9%. Among outdoor allergens, 31.6% (n=6) was grass mix, followed by 15.8% (n=3) mugwort, 15.8% (n=3) cat, 10.5% (n=2) horse, 10.5% (n=2) birch, 10.5% (n=2) *Alternaria alternata* and 5.3% (n=1) *Cladosporium herbarum*.

In the study conducted by Meher BK et al 2021 reported Kentucky bluegrass, *Dermatophagoides pteronyssinus, Dermatophagoides farinae,* Timothy grass and Alternaria alternans in 25%, 22.5%, 21.3%, 20% and 20% of the study participants as the common aeroallergens.[(88)]

In a study conducted to study the fungi growth in indoors in Delhi by Sharma R et al 2011 reported different species of Aspergillus, Alternaria, Cladosporium and Penicillium constituting 88.6% of the colonies.[(90)]

Rice grain dust (31%) and cockroach (18.3%) were the common aeroallergens reported in the study conducted by Raj D et al 2013.[(10)]

Limitations of the study-The study was conducted as a cross-sectional study design, which limits the ability to establish the causal relationship between aeroallergen and development of respiratory disease. The study was conducted as a single-centre study and tertiary care setting; hence, the results may not be generalized in other region or settings.

CONCLUSION

This study examined aeroallergen sensitization in children with allergic respiratory diseases, focusing on those between early childhood and late adolescence. The majority of participants were boys, with common cold being the most frequent clinical presentation. Allergic rhinitis was the most prevalent condition, followed by bronchial asthma and united airway disease. Many children were sensitized to one or more allergens with house dust mites, particularly Dermatophagoides pteronyssinus, being the most frequently identified aeroallergen in polysensitized individuals. House dust mites which included Dermatophagoides pteronyssinus and Dermatophagoides farinae were the primary aeroallergens linked to severe disease outcomes across all respiratory conditions in this particular study.

SUMMARY

A one-year cross-sectional study in children with allergic respiratory disease was conducted at KAHER'S Dr. Prabhakar Kore hospital, Belgaum-590010, Karnataka aimed to assess the sensitization profile of children in the age group 5-18 years with allergic respiratory diseases at a tertiary care centre and to identify the most prevalent aero-allergens that might result in severe disease outcome.

Given the increasing prevalence of allergic conditions in urban areas, understanding the sensitization profile in children is crucial for early diagnosis and targeted management. House dust mites, particularly Dermatophagoides pteronyssinus and Dermatophagoides farinae, were the predominant allergens associated with allergic rhinitis, bronchial asthma, and united airway disease. The study highlights the importance of recognizing common indoor and outdoor allergens, which can guide effective preventive strategies and therapeutic interventions for affected children.

The significance of this research lies in its potential to improve clinical outcomes by emphasizing the need for routine allergen screening in pediatric populations. Identifying specific sensitization patterns allows for personalized treatment approaches, reducing disease severity and improving quality of life. Early intervention and environmental control measures can help mitigate allergic symptoms and prevent disease progression. This study underscores the necessity of implementing preventive strategies, particularly in urban settings where exposure to allergens is high. Future research should focus on long-term monitoring of sensitization patterns and evaluating the effectiveness of targeted interventions in managing allergic respiratory diseases in children.

BIBLIOGRAPHY

1. Ownby D. Environmental factors versus genetic determinants of childhood inhalant allergies. Journal of Allergy and Clinical Immunology. 1990 Sep;86(3):279–87.
2. Burks AW, Jones SM, Boyce JA, Sicherer SH, Wood RA, Assa'ad A, et al. NIAID-Sponsored 2010 Guidelines for Managing Food Allergy: Applications in the Pediatric Population. Pediatrics. 2011 Oct 10;128(5):955–65.
3. Branum AM, Lukacs SL. Food Allergy Among Children in the United States. PEDIATRICS. 2009 Nov 16;124(6):1549–55.
4. Pawankar R, Canonica GW, Holgate ST, Lockey RF. Allergic diseases and asthma. Current Opinion in Allergy & Clinical Immunology. 2012 Feb;12(1):39–41.
5. Leung DYM, Guttman-Yassky E. Deciphering the complexities of atopic dermatitis: shifting paradigms in treatment approaches. The Journal of Allergy and Clinical Immunology [Internet]. 2014 Oct 1;134(4):769–79. Available from: https://www.ncbi.nlm.nih.gov/pubmed/25282559
6. Saha GK. House dust mite allergy in Calcutta, India: evaluation by RAST. Annals of allergy [Internet]. 1993 Apr;70(4):305–9. Available from: https://pubmed.ncbi.nlm.nih.gov/7682041/
7. Mahesh PA, Kummeling I, Amrutha DH, Vedanthan PK. Effect of Area of Residence on Patterns of Aeroallergen Sensitization in Atopic Patients. American Journal of Rhinology & Allergy. 2010 Sep;24(5):e98–103.
8. Trakultivakorn M, Sangsupawanich P, Vichyanond P. Time Trends of the Prevalence of Asthma, Rhinitis and Eczema in Thai Children–ISAAC

(International Study of Asthma and Allergies in Childhood) Phase Three. Journal of Asthma. 2007 Jan;44(8):609–11.

9. Serebrisky D, Wiznia A. Pediatric Asthma: A Global Epidemic. Annals of Global Health [Internet]. 2019 Jan 22;85(1). Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7052318/
10. Raj D, Lodha R, Pandey A, Mukherjee A, Agrawal A, Kabra SK. Aeroallergen sensitization in childhood asthmatics in Northern India. Indian Pediatrics. 2013 Jul 5;50(12):1113–8.
11. Ciprandi G, Caimmi D, Giudice MM del, Rosa ML, Salpietro C, Marseglia GL. Recent Developments in United Airways Disease. Allergy, Asthma & Immunology Research [Internet]. 2012 Feb 8 [cited 2022 May 15];4(4):171–7. Available from: https://synapse.koreamed.org/articles/1052264
12. Mishra V, Mahmood T, Mishra J. Identification of common allergens for united airway disease by skin prick test. Indian Journal of Allergy Asthma and Immunology. 2016 Jan 1;30(2):76–6.
13. Golden DBK. Insect Sting Anaphylaxis. Immunology and Allergy Clinics of North America. 2007 May;27(2):261–72.
14. F.M. Kniest, Young E, Praag V, Vos HS, Kort H, Koers WJ, et al. Clinical evaluation of a double-blind dust-mite avoidance trial with mite-allergic rhinitic patients. Clinical & Experimental Allergy. 1991 Jan 1;21(1):39–47.
15. Luczynska CM, Li Y, Chapman MD, Platts-Mills TAE. Airborne Concentrations and Particle Size Distribution of Allergen Derived from Domestic Cats (Felis domesticus): Measurements Using Cascade Impactor, Liquid Impinger, and a Two-site Monoclonal Antibody Assay forFel dI. American Review of Respiratory Disease. 1990 Feb;141(2):361–7.

16. Burger HC. Bioaerosols: prevalence and health effects in the indoor environment. The Journal of Allergy and Clinical Immunology. 1990 Nov 1;86(5):687–701.
17. Pedreira FA, Guandolo VL, Feroli EJ, Mella GW, Weiss IP. Involuntary smoking and incidence of respiratory illness during the first year of life. Pediatrics [Internet]. 1985 Mar;75(3):594–7. Available from: https://pubmed.ncbi.nlm.nih.gov/3975130/
18. Shamji MH, Valenta R, Jardetzky T, Verhasselt V, Durham SR, Würtzen PA, et al. The role of allergen-specific IgE, IgG and IgA in allergic disease. Allergy. 2021 Jun 8;76(12).
19. Frati F, Incorvaia C, Cavaliere C, Di Cara G, Marcucci F, Esposito S, et al. The skin prick test. Journal of Biological Regulators and Homeostatic Agents [Internet]. 2018 Jan 1;32(1 Suppl. 1):19–24. Available from: https://pubmed.ncbi.nlm.nih.gov/29552869/
20. Vos T, Lim SS, Abbafati C, Abbas KM, Abbasi M, Abbasifard M, et al. Global Burden of 369 Diseases and Injuries in 204 Countries and territories, 1990–2019: a Systematic Analysis for the Global Burden of Disease Study 2019. The Lancet. 2020 Oct 17;396(10258):1204–22.
21. Baard CB, Franckling-Smith Z, Munro J, Workman L, Zar HJ. Asthma in South African adolescents: a time trend and risk factor analysis over two decades. ERJ Open Research [Internet]. 2021 Apr 1;7(2). Available from: https://openres.ersjournals.com/content/7/2/00576-2020
22. Asher MI, Keil U, Anderson HR, Beasley R, Crane J, Martinez F, et al. International study of asthma and allergies in childhood (ISAAC): rationale and methods. European Respiratory Journal [Internet]. 1995 Mar 1;8(3):483–91. Available from: https://erj.ersjournals.com/content/erj/8/3/483.full.pdf

23. Gupta S, Daniel R, Aggarwal P, Kalaivani M. Prevalence of asthma among children in India: A systematic review and meta-analysis. Lung India. 2022;39(4):357.
24. Dahl R, Andersen PS, Chivato T, Valovirta E, de Monchy J. National prevalence of respiratory allergic disorders. Respiratory Medicine [Internet]. 2004 May 1 [cited 2022 Oct 9];98(5):398–403. Available from: https://www.sciencedirect.com/science/article/pii/S0954611103004189
25. Singh AB, Kumar P. Aeroallergens in clinical practice of allergy in India. An overview. Annals of agricultural and environmental medicine : AAEM [Internet]. 2003;10(2):131–6. Available from: https://pubmed.ncbi.nlm.nih.gov/14677902/
26. Rabe A, Wei Jie Loke, Khushboo Gurjar, Brackley A, Don Eliseo Lucero-Prisno. Global Burden of Asthma, and Its Impact on Specific Subgroups: Nasal Polyps, Allergic Rhinitis, Severe Asthma, Eosinophilic Asthma. Journal of Asthma and Allergy [Internet]. 2023 Oct 1;Volume 16:1097–113. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10563777/#:~:text=Globally%2C%20of%20those%20with%20asthma
27. Smith JG, Disney ME, Williams JC, Goels ZA. Clinical Significance of Skin Reactions to Mite Extracts in Children with Asthma. Br Med J . 1969 Jun 21;2(5659):723–6.
28. Crater DD, Heise S, Perzanowski M, Herbert R, Morse CG, Hulsey TC, et al. Asthma Hospitalization Trends in Charleston, South Carolina, 1956 to 1997: Twenty-fold Increase Among Black Children During a 30-Year Period. Pediatrics. 2001 Dec 1;108(6):e97–7.

29. Pomés A, Chapman MD, Wünschmann S. Indoor Allergens and Allergic Respiratory Disease. Current Allergy and Asthma Reports. 2016 May 16;16(6).
30. Singh M, Hays A. Indoor and Outdoor Allergies. Primary Care: Clinics in Office Practice. 2016 Sep;43(3):451–63.
31. Min YG. The Pathophysiology, Diagnosis and Treatment of Allergic Rhinitis. Allergy, Asthma and Immunology Research [Internet]. 2010;2(2):65. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2846743/
32. Pawankar R. Allergic diseases and asthma: a global public health concern and a call to action. World Allergy Organization Journal. 2014;7(1):12.
33. Barne M, Singh S, Mangal DK, Singh M, Awasthi S, Mahesh PA, et al. Global Asthma Network Phase I, India: Results for allergic rhinitis and eczema in 127,309 children and adults. Journal of Allergy and Clinical Immunology: Global [Internet]. 2022 May 1 [cited 2022 Oct 19];1(2):51–60. Available from: https://www.sciencedirect.com/science/article/pii/S2772829322000133#:~:text=The%20ISAAC%20phase%20III%20studied
34. Blomme K, Tomassen P, Lapeere H, Huvenne W, Bonny M, Acke F, et al. Prevalence of Allergic Sensitization versus Allergic Rhinitis Symptoms in an Unselected Population. International Archives of Allergy and Immunology [Internet]. 2013;160(2):200–7. Available from: https://www.karger.com/Article/Abstract/339853
35. Zuberbier T, Lötvall J, Simoens S, Subramanian SV, Church MK. Economic burden of inadequate management of allergic diseases in the European Union: a GA2LEN review. Allergy. 2014 Aug 1;69(10):1275–9.
36. Tidke M, Borghare PT, Pardhekar P, Nasre Y, Gomase K, Chaudhary M. Recent Advances in Allergic Rhinitis: A Narrative Review. Cureus [Internet].

2024 Sep 4 [cited 2024 Nov 30]; Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11450520/

37. Ukena D, Fishman L, Niebling WB. Bronchial Asthma. Deutsches Aerzteblatt Online [Internet]. 2008 May 23;105(21). Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2696883/
38. Mortimer K, Lesosky M, García-Marcos L, Innes Asher M, Pearce N, Ellwood E, et al. The burden of asthma, hay fever and eczema in adults in 17 countries: GAN Phase I study. European Respiratory Journal. 2022 Feb 24;2102865.
39. Moore WC, Meyers DA, Wenzel SE, Teague WG, Li H, Li X, et al. Identification of Asthma Phenotypes Using Cluster Analysis in the Severe Asthma Research Program. American Journal of Respiratory and Critical Care Medicine [Internet]. 2010 Feb 15;181(4):315–23. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2822971/
40. National Asthma Education and Prevention Program. Section 2, Definition, Pathophysiology and Pathogenesis of Asthma, and Natural History of Asthma [Internet]. Nih.gov. National Heart, Lung, and Blood Institute (US); 2007. Available from: https://www.ncbi.nlm.nih.gov/books/NBK7223/
41. Reddel H, Ware S, Marks G, Salome C, Jenkins C, Woolcock A. Differences between asthma exacerbations and poor asthma control. The Lancet. 1999 Jan;353(9150):364–9.
42. Lizzo JM, Cortes S. Pediatric asthma [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK551631/
43. Rimmer J, Ruhno J. 6: Rhinitis and asthma: united airway disease. The Medical Journal of Australia. 2006 Nov 1;185(10):565–71.

44. Fokkens W, Reitsma S. Unified Airway Disease. Otolaryngologic Clinics of North America. 2023 Feb;56(1):1–10.
45. Klain A, Cristiana Indolfi, Dinardo G, Licari A, Cardinale F, Caffarelli C, et al. United airway disease. PubMed. 2021 Nov 29;92(S7):e2021526–6.
46. de Benedictis FM, del Giudice MMiraglia, Severini S, Bonifazi F. Rhinitis, sinusitis and asthma: one linked airway disease. Paediatric Respiratory Reviews. 2001 Dec;2(4):358–64.
47. Biagtan M, Viswanathan R, Bush RK. Immunotherapy for House Dust Mite Sensitivity: Where Are the Knowledge Gaps? Current Allergy and Asthma Reports. 2014 Oct 30;14(12).
48. Miglani A, Lal D, Divekar RD. Unified Airway Disease: Diagnosis and Subtyping. Otolaryngologic clinics of North America [Internet]. 2023 Feb;56(1):11–22. Available from: https://pubmed.ncbi.nlm.nih.gov/36410988/
49. Meena RS, Meena D, Yogesh Aseri, Singh BK, Verma PC. Chronic Rhino-Sinusitis and Asthma: Concept of Unified Airway Disease (UAD) and its Impact in Otolaryngology. Indian Journal of Otolaryngology and Head & Neck Surgery. 2012 Feb 1;65(S2):338–42.
50. Larrabee YC, Reisacher W. Intradermal testing after negative skin prick testing for patients with high suspicion of allergy. International Forum of Allergy & Rhinology. 2015 Mar 10;5(6):547–50.
51. Krzych-Fałta E, Furmańczyk K, Dziewa-Dawidczyk D, Wojas O, Reklewska K, Samoliński BK. The role of the nasal allergen provocation test in local allergic rhinitis cases: a preliminary report. Advances in Dermatology and Allergology. 2020;37(6):890–7.

52. van Hage M, Hamsten C, Valenta R. ImmunoCAP assays: Pros and cons in allergology. Journal of Allergy and Clinical Immunology. 2017 Oct;140(4):974–7.
53. Johansson S. ImmunoCAP® Specific IgE test: an objective tool for research and routine allergy diagnosis. Expert Review of Molecular Diagnostics. 2004 May;4(3):273–9.
54. Alhajj M, Farhana A, Zubair M. Enzyme Linked Immunosorbent Assay (ELISA) [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK555922/
55. Rashmi B, Patil S, Sindhu B, Patil S. Pediatric asthma: Prevalence and socio-cultural factors affecting asthma management in a rural area of Northern Karnataka. Indian Journal of Community Medicine. 2021;46(1):24.
56. Lopez M, Jaggi V, Dalal A, Ramesh B, Tikkiwal S, Chaudhry A, et al. Coexistence of allergic rhinitis and asthma in Indian patients: The CARAS survey. Lung India. 2019;36(5):411.
57. Acevedo-Prado A, Seoane-Pillado T, López-Silvarrey-Varela A, Salgado FJ, Cruz MJ, Faraldo-Garcia A, et al. Association of rhinitis with asthma prevalence and severity. Scientific Reports. 2022 Apr 16;12(1).
58. Shikha G, Swami H. Trends of sensitization pattern to aeroallergens among the patients with allergic rhinitis and/or bronchial asthma in Bangalore: A cross sectional study. Medical Journal Armed Forces India. 2021 Feb;
59. Stevenson MD, Sellins S, Grube E, Schroer K, Gupta J, Wang N, et al. Aeroallergen Sensitization in Healthy Children: Racial and Socioeconomic Correlates. The Journal of Pediatrics [Internet]. 2007 Aug 1 [cited 2022 Dec 18];151(2):187–91. Available from:

https://www.sciencedirect.com/science/article/abs/pii/S0022347607002375?via%3Dihub

60. Moitra S, Mahesh PA, Moitra S. Allergic rhinitis in India. Clinical & Experimental Allergy. 2023 Mar;
61. Pacheco CM, Ciaccio CE, Nazir N, Daley CM, DiDonna A, Choi WS, et al. Homes of low-income minority families with asthmatic children have increased condition issues. Allergy and Asthma Proceedings [Internet]. 2014 Nov 1;35(6):467–74. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4210655/
62. Chooniedass R, Soller L, Hsu E, To S, Cameron SB, Chan ES. Parents of children with food allergy. Annals of Allergy, Asthma & Immunology. 2020 Dec;125(6):674–9.
63. Umanets TR, Buratynska AA, Rudnev OO, Lapshyn VF, Antipkin YuG. Sensitization profile in children with allergic respiratory diseases. Asthma and allergy. 2020;(3):29–34.
64. Feliu A, González-de-Olano D, González E, Rodriguez B, Ruiz-Hornillos J, Jimeno L, et al. A multicenter study of sensitization profiles in an allergic pediatric population in an area with high allergen exposure. Journal of investigational allergology & clinical immunology [Internet]. 2013;23(5):337–44. Available from: https://pubmed.ncbi.nlm.nih.gov/24260979/
65. Dai L, Liu J, Zhao Q, Li M, Zhou Y, Chen Z, et al. Investigation of Allergic Sensitizations in Children With Allergic Rhinitis and/or Asthma. Frontiers in Pediatrics. 2022 Mar 15;10.
66. Bhagyashree P, Kumar P, Patavardhan P, MS V. Allergic Sensitization in Asthmatic Children Attending Tertiary Care Center in Western Rajasthan. European Journal of Rhinology and Allergy [Internet]. 2023 Sep 1;6(2):55–60.

Available from: https://www.eurjrhinol.org/Content/files/sayilar/109/EJRA_August_2023-55-60.pdf

67. Kumar D, Anand M, Sharma V, Goyal R, Gupta N, Gupta D. Allergen sensitivity profile among symptomatic children: A descriptive study from North India. Indian Journal of Allergy Asthma and Immunology. 2022 Jul 1;36(2):85–91.
68. Srivastava S, Chauhan K, Prasad R. Sensitization to indoor allergens in children with bronchial asthma. Indian Journal of Allergy, Asthma and Immunology. 2020;34(1):39.
69. Lazova S, Baleva M, Priftis S, Naseva E, Velikova T. Atopic Status in Children with Asthma and Respiratory Allergies—Comparative Analysis of Total IgE, ImmunoCAP Phadiatop/fx5 and Euroimmun Pediatric Immunoblot. Sinusitis [Internet]. 2022 Jun 1;6(1):1–14. Available from: https://www.mdpi.com/2673-351X/6/1/1
70. Ying X, Qi X, Yin Y, Wang H, Zhang H, Jiang H, et al. Allergens sensitization among children with allergic diseases in Shanghai, China: age and sex difference. Respiratory Research. 2022 Apr 15;23(1).
71. Yan Y, Xu Y, Zheng Q, Shi Guo Y. Asian Pacific Journal of Allergy and Immunology The prevalence and sex difference of allergen sensitization among adult patients with allergic diseases in Shanghai, China. Asian Pac J Allergy Immunol [Internet]. 2019 [cited 2023 Dec 8]; Available from: https://www.apjai-journal.org/wp-content/uploads/2019/09/4.pdf
72. Liu T, Lai S, Li W, Jiang Y. Prevalence of food allergen and aeroallergen sensitization among children in Sichuan province. Medicine [Internet]. 2020 Jun

29 [cited 2025 Mar 12];99(27):e21055–5. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7337612/

73. Gezmu AM, Kung SJ, Jemal Zeberga Shifa, Nakstad B, Brooks M, Joel D, et al. Pediatric Spectrum of Allergic Diseases and Asthma in a Tertiary Level Hospital in Botswana: an Exploratory Retrospective Cross-Sectional Study. Journal of Asthma and Allergy [Internet]. 2020 Jun 30 [cited 2025 Mar 12];Volume 13:213–23. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7342389/
74. McColley SA, Carroll JL, Curtis S, Loughlin GM, Sampson HA. High Prevalence of Allergic Sensitization in Children With Habitual Snoring and Obstructive Sleep Apnea. Chest. 1997 Jan;111(1):170–3.
75. Sporik R, Holgate ST, Platts-Mills TAE, Cogswell JJ. Exposure to House-Dust Mite Allergen (Der pI) and the Development of Asthma in Childhood. New England Journal of Medicine. 1990 Aug 23;323(8):502–7.
76. Langley SJ, Goldthorpe S, Craven M, Morris J, Woodcock A, Custovic A. Exposure and sensitization to indoor allergens: Association with lung function, bronchial reactivity, and exhaled nitric oxide measures in asthma. Journal of Allergy and Clinical Immunology. 2003 Aug;112(2):362–8.
77. Sala KA, Carroll CL, Tang YS, Aglio T, Dressler AM, Schramm CM. Factors Associated with the Development of Severe Asthma Exacerbations in Children. Journal of Asthma. 2011 Jun 6;48(6):558–64.
78. Arshad SH, Tariq SM, Matthews S, Hakim E. Sensitization to Common Allergens and Its Association With Allergic Disorders at Age 4 Years: A Whole Population Birth Cohort Study. PEDIATRICS. 2001 Aug 1;108(2):e33–3.

79. Illi S, von Mutius E, Lau S, Niggemann B, Grüber C, Wahn U. Perennial allergen sensitisation early in life and chronic asthma in children: a birth cohort study. The Lancet. 2006 Aug;368(9537):763–70.
80. Roberts G, Zhang H, Karmaus W, Raza A, Scott M, Matthews S, et al. Trends in cutaneous sensitization in the first 18 years of life: results from the 1989 Isle of Wight birth cohort study. Clinical & Experimental Allergy. 2012 Sep 21;42(10):1501–9.
81. Sattar HA, Mobayed H, al-Mohammed AA, Ibrahim AS, Jufairi AA, Balamurugan P, et al. The pattern of indoor and outdoor respiratory allergens in asthmatic adult patients in a humid and desert newly developed country. European annals of allergy and clinical immunology [Internet]. 2003 Oct;35(8):300–5. Available from: https://pubmed.ncbi.nlm.nih.gov/14653049/
82. Janahi IA, Bener A, Bush A. Prevalence of Asthma among Qatari Schoolchildren: International Study of Asthma and Allergies in Childhood, Qatar. Pediatric Pulmonology. 2005;41(1):80–6.
83. Sharif H, Salih E, Awad H, Albarazi R. Common aeroallergens among children with allergies in the United Arab Emirates. Indian Journal of Allergy, Asthma and Immunology. 2018;32(1):4.
84. Al-Rabia M. Prevalence and severity of sensitization reaction to food and inhalant allergens among allergic patients from Jeddah city: Saudi Arabia. International Journal of Community Medicine and Public Health [Internet]. 2016 Jan 1 [cited 2025 Mar 12];2908–15. Available from: https://www.ijcmph.com/index.php/ijcmph/article/view/263

85. AlKhater SA. Sensitization to Common Aeroallergens in Asthmatic Children in the Eastern Region of Saudi Arabia. Saudi Journal of Medicine & Medical Sciences [Internet]. 2017 [cited 2023 Dec 30];5(2):136–41. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6298380/
86. Zahraldin K, Chandra P, Tuffaha A, Ehlayel M. Sensitization to Common Allergens Among Children with Asthma and Allergic Rhinitis in Qatar. Journal of Asthma and Allergy. 2021 Mar;Volume 14:287–92.
87. Yadav S, Mathur M, Agarwal G, Sharma AK. A Descriptive Study on Aeroallergen Sensitivity Pattern in Children with Bronchial Asthma and/or Allergic Rhinitis. Journal of Child Science. 2020 Jan;10(01):e48–52.
88. Meher BK, Pradhan DD, Mahar J, Sahu SK. Prevalence of Allergic Sensitization in Childhood Asthma. Cureus. 2021 May 29;
89. Uysal P, Erge D, Yenigün A. Clinical Characteristics of Fungal Sensitization in Children with Allergic Respiratory Diseases. Meandros Medical and Dental Journal. 2016 Aug 1;17(2):74–82.
90. Sharma R, Deval R, Priyadarshi V, Gaur SN, Singh VP, Singh AB. Indoor Fungal Concentration in the Homes of Allergic/Asthmatic Children in Delhi, India. Allergy & Rhinology. 2011 Jan;2(1):ar.2011.2.0005.

ANNEXURES

ANNEXURE – I - INFORMED CONSENT FORM

"AEROALLERGEN SENSITIZATION PROFILE IN CHILDREN WITH ALLERGIC RESPIRATORY DISEASES BY EAST (ENZYME ALLERGO SORBENT TEST) METHOD IN THE AGE 5-18 YEARS : A ONE YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE CENTRE"

Name of Student/Principal Investigator:_____

Name of Guide/Co Investigators: _____

Introduction: To assess the sensitization profile of children in the age group 5-18 years with allergic respiratory diseases at a tertiary care centre Explanation of procedure: The study requires a plain blood sample to be collected and tested for allergen sensitization profile in children.

Withdrawal from participation in the study: Your child's participation in this study is your voluntary decision. Whether to participate or not to participate will not affect your current or future relationship with KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

You are free to discontinue the participation in the study at any time for any reasons and you will not be paid any reimbursement for participation in the research 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.

Possible benefits from participating in the study: The child will not have any benefits by participating in this study. 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 Dr.Sumanth Pothineni 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:

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

INFORMED CONSENT STATEMENT

I hereby agree for my child's participation in this study and am making a voluntary decision to participate in the study **" AEROALLERGEN SENSITIZATION PROFILE IN CHILDREN WITH ALLERGIC RESPIRATORY DISEASES BY EAST(ENZYME ALLERGO SORBENT TEST) METHOD IN THE AGE 5-18 YEARS: A ONE YEAR CROSS SECTIONAL STUDY AT A TERTIARY CARE CENTRE".** My signature below indicates that I/we parents of the child have agreed that my child will participate in the study 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:

Date:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator

Signature of the investigator:

ANNEXURE II: PROFORMA

NAME-

AGE/SEX -

CHIEF COMPLAINTS-

PRECIPITATING FACTORS/MODIFYING FACTORS-

INHALANTS- DRUG ALLERGY-

IRRITANTS- WORK/SCHOOL-

INFECTIONS- STRESS-

EXERCISE- PHYSICAL FACTORS -

RADIOCONTRAST MEDIA- HYMENOPTERA-

OTHERS-

ENVIRONMENTAL HISTORY-

LOCATION- BASEMENT- CARPETS-

HOUSE- PETS- MATTRESS-

BEDROOM- SMOKE- PILLOW-

PLANTS- OTHERS-

PAST MEDICAL HISTORY-

H/O TOBACCO EXPOSURE-

PERSONAL H/O ANAPHYLAXIS-

H/O FOOD ALLERGY

H/O DRUG ALLERGY-

H/O INSECT ALLERGY-

H/O LATEX ALLERGY-

H/O RADIOCONTRAST MEDIA ALLERGY-

NONE OF THE ABOVE-

MEDICATIONS AND ILLNESSES-

HOSPITALIZATIONS/OPERATIONS-

FAMILY HISTORY-(ALLERGY/ASTHMA/OTHER)

BIRTH AND DEVELOPMENT HISTORY-

FEEDING/DIETIC HISTORY-

IMMUNIZATION HISTORY-

PHYSICAL EXAMINATION-

Vital Signs- HR-

RR-

BP-

TEMP-

GENERAL APPEARANCE- NORMAL/RESPIRATORY DISTRESS

HEAD TO TOE-

HEAD AND FACE- NORMAL/SINUS TENDERNESS

EYES-CONJUNCTIVA/EYELIDS-

NORMAL/SHINERS/COBBLESTONING/DISCHARGE

EARS-

EXTERNAL CANALS- NORMAL / CERUMEN

TYMPANIC MEMBRANE - NORMAL / CONGESTED / RETRACTED

EUSTACHIAN TUBES- DISCHARGE/ EFFUSION

NOSE-

MUCOSA- NORMAL/ BOGGY/ PALE/ POLYPS/ NONE

ERYTHEMA / CREASE/

RHINORRHEA- NONE/ CLEAR/ MUCOID/ PURULENT

TURBINATES- OPEN/ PARTIALLY OBSTRUCTED / TOTALLY OBSTRUCTED

SEPTUM - NORMAL / DEVIATED

TEETH AND GUMS - NORMAL / MALOCCLUSION/ POOR DENTITION/

GINGIVITIS

OROPHARYNX- NORMAL / INJECTED/ COBBLE STONING / EXUDATES/

ENLARGED TONSILS

THYROID - NORMAL / NODULES / THYROMEGALY

RESPIRATORY

EFFORT- NORMAL / RETRACTIONS/ ACCESSORY MUSCLE USE

AUSCULTATION - CLEAR/ WHEEZE/ CRACKLE

CARDIOVASCULAR -

AUSCULTATION-

MURMUR -

PERIPHERAL PULSES-

CLUBBING -

GASTROINTESTINAL -

NORMAL / TENDER/ MASS

LYMPHATIC - CERVICAL- NORMAL/ ABNORMAL

AXILLARY - NORMAL/ABNORMAL

PROVISIONAL DIAGNOSIS -

BRONCHIAL ASTHMA- WELL CONTROLLED/ PARTLY/ UNCONTROLLED

ALLERGIC RHINITIS- INTERMITTENT / PERSISTENT

INVESTIGATIONS -

PULMONARY FUNCTION TEST -

EAST (ENZYME ALLERGO SORBENT TEST) METHOD

OTHERS -

FINAL IMPRESSION-

ADVICE -

MDI

ORAL MEDICATION-

ENVIRONMENTAL CONTROL MEASURES-

ANNEXURE III: MASTERCHART

S.NO	NAME	AGE	SEX	CLINICAL SYMPTOMS	PRECIPITATING FACTORS/TRIGGERS	CLINICAL PRESENTATION	ALLERGEN SENSITIVITY	CLASS	DIAGNOSIS	TREATMENT	ACUTE EXACERBATIONS	GEOGRAPHIC DISTRIBUTION
1)	Siddhappa	15 years	M	Cough+cold and fever	Not known	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 2 - Daily Low dose ICS	Nil in last 1 year	Rural
2)	Samarth	6 years	M	Cough+cold and fever	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
3)	Jenil Hitesh Patil	7 years	M	Cough+cold and fever	House dust	Bronchial Asthma+ Allergic rhinitis	Nil	Nil	Moderate bronchial asthma + mild intermittent Allergic rhinitis	Step 3- Daily low dose ICS-LABA	Nil in last 1 year	Urban
4)	Mohammed Azlan	5 years	M	Cough+cold+noisy breathing	House dust	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 2 - Daily Low dose ICS	Nil in last 1 year	Urban
5)	Kushi	6 years	F	Cough+cold+noisy breathing	Not known	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 1- Low dose ICS	Nil in last 1 year	Urban
6)	Suman	14 years	F	Cough+cold and breathing difficulty	House dust+exercise	Bronchial Asthma	Der. pteronyssinus	Class 2	Moderate bronchial asthma	Step 3 - medium dose ICS-LABA	1 in last 1 year	Urban
							Der. farinae	Class 2				
7)	Harshit	6 years	M	Fever+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
8)	Satvik	7 years	M	Fever+running nose+noisy breathing	Not known	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 2 - Daily Low dose ICS	Nil in last 1 year	Urban
9)	Richa	6 years	F	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
10)	Pranjal	6 years	F	Fever+running nose	Not known	Allergic rhinitis	Horse	Class 1	Moderate to severe intermittent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	1 in last 1 year	Rural
11)	Roselin	13 years	F	Cough+ sore throat	House dust+pet allergy	Allergic rhinitis	Cat	Class 2	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
12)	Virat	5 years	M	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban

13)	Yash	8 years	M	Cough+cold and fever	House dust	Bronchial Asthma	Grass mix 2	Class 1	Mild bronchial asthma	Step 2 - Daily Low dose ICS	Nil in last 1 year	Urban
14)	Pallavi	12 years	F	Running nose on and off	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
15)	Poornima	14 years	F	Cough+cold+running nose	House dust	Allergic rhinitis	Nil	Nil	Mild persistent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	Nil in last 1 year	Rural
16)	Sultan	10 years	M	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
17)	Gagangouda	14 years	M	Cough+cold+noisy breathing	Not known	Bronchial Asthma	Nil	Nil	Moderate bronchial asthma	Step 3 - Daily low dose ICS-LABA	Nil in last 1 year	Rural
18)	Prasanna	8 years	M	Fever+running nose	House dust	Bronchial Asthma+ Allergic rhinitis	Der. pteronyssinus	Class 5	Severe bronchial asthma + mild persistent allergic rhinitis	Step 4 - high dose ICS-LABA	2 in last 1 year	Urban
							Der. farinae	Class 5				
19)	Pranav	11 years	M	Cough+cold and fever	House dust and pollen	Allergic rhinitis	Der. pteronyssinus	Class 3	Moderate to severe intermittent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	1 in last 1 year	Urban
							Der. farinae	Class 2				
20)	Abhinav	6 years	M	Cough+cold and breathing difficulty	Cold weather	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
21)	Manikant	9 years	M	Fever+running nose	house dust	Allergic rhinitis	Der. pteronyssinus	Class 3	Mild persistent allergic rhinitis	Oral antihistamines	1 in last 1 year	Urban
							Der. farinae	Class 1				
22)	Ayesha	8 years	F	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
23)	Tanisha	6 years	F	Running nose on and off	Not known	Allergic rhinitis	Der. pteronyssinus	Class 1	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
24)	Ruthvik	9 years	M	Fever+running nose	House dust	Allergic rhinitis	Der. pteronyssinus	Class 4	Moderate to severe persistent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	2 in last 1 year	Urban
							Der. farinae	Class 3				

25)	Vikrant	7 years	M	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
26)	Deepak	14 years	M	Cough+cold+running nose	Stress+ excerise, pollen dust	Bronchial Asthma+ Allergic rhinitis	Mugwort	Class 3	Moderate bronchial asthma + moderate to severe persistent Allergic rhinitis	Step 3 - medium dose ICS-LABA	3 in last 1 year	Urban
							Der. pteronyssinus	Class 4				
							Der. farinae	Class 4				
27)	Aisha	6 years	F	Cough+cold on and off	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
28)	Shlok prashant	12 years	M	Running nose on and off	House dust	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
29)	Swagat kumar	6 years	M	Cough+cold+noisy breathing	House dust	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
30)	Shreyas	11 years	M	Cough+cold+running nose	House dust	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 1- Low dose ICS	Nil in last 1 year	Urban
31)	Tarun	7 years	M	Cough+cold+noisy breathing	Not known	Bronchial Asthma+ Allergic rhinitis	Nil	Nil	Mild bronchial asthma + mild persistent allergic rhinitis	Step 2 - Daily Low dose ICS + oral antihistamines	Nil in last 1 year	Urban
32)	Zulfakar	5 years	M	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
33)	Sanket	10 years	M	Fever+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
34)	Sneha	8 years	F	Cough+cold+running nose	House dust	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
35)	Vishwanath	6 years	M	Cough+nasal congestion+ breathing difficulty	House dust	Bronchial Asthma	Der. pteronyssinus	Class 4	Severe bronchial asthma	Step 4 - high dose ICS-LABA	2 in last 1 year	Urban
							Der. farinae	Class 5				
							Cat	Class 1				
36)	Devish	7 years	M	Cough+cold and fever	Cold weather	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
37)	Roshan	6 years	M	Running nose on and off	House dust	Allergic rhinitis	Der. pteronyssinus	Class 4	Moderate to severe persistent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	3 in last 1 year	Urban
							Der. farinae	Class 5				

							Horse	Class 2				
38)	Amey	8 years	M	Cough+cold+running nose	Cold weather	Bronchial Asthma	Der. pteronyssinus	Class 2	Mild bronchial asthma	Step 2 - Daily Low dose ICS	Nil in last 1 year	Urban
							Der. farinae	Class 2				
39)	Dhruv Metri	10 years	M	Fever+running nose	Not known	Allergic rhinitis	Horse	Class 1	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
40)	Shravani	15 years	F	Cough+cold+running nose	Cold weather	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 1- Low dose ICS	Nil in last 1 year	Rural
41)	Jayaraj	13 years	M	Cold+noisy breathing	House dust	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
42)	Shivraj	12 years	M	Cough+cold on and off	House dust and pollen	Bronchial Asthma	Grass mix 2	Class 1	Mild bronchial asthma	Step 2 - Daily Low dose ICS	1 in last 1 year	Urban
43)	Aditya	8 years	M	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
44)	Narayan	11 years	M	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
45)	Meha	7 years	F	Cough+cold+running nose	House dust and pollen	Allergic rhinitis	Der. pteronyssinus	Class 3	Moderate to severe persistent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	2 in last 1 year	Rural
							Der. farinae	Class 4				
46)	Mohammed Ayan	6 years	M	Cough+cold and fever	House dust and pollen	Allergic rhinitis	Grass mix 2	Class 5	Moderate to severe persistent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	2 in last 1 year	Urban
							Birch	Class 1				
							Mugwort	Class 2				
							Der. pteronyssinus	Class 1				
							Der. farinae	Class 1				
47)	Bhusit	6 years	M	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
48)	Kushal Gopal	8 years	M	Cough+cold and breathing difficulty	Cold weather	Bronchial Asthma	Der. pteronyssinus	Class 4	Moderate bronchial asthma	Step 3 - Daily low dose ICS-LABA	2 in last 1 year	Rural
							Der. farinae	Class 5				

49)	Aditya G	7 years	M	Cough+cold and fever	Grass+Pollen exposure	Bronchial Asthma	Grass mix 2	Class 1	Mild bronchial asthma	Step 1- Low dose ICS	Nil in last 1 year	Rural
							Cladosporium herbarum	Class 1				
							Alternaria alternata	Class 1				
50)	Maitrika	9 years	F	Cough+cold+running nose	House dust and pollen	Allergic rhinitis	Alternaria alternata	Class 1	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
51)	Rachita	7 years	F	Fever+running nose+noisy breathing	House dust	Bronchial Asthma	Der. pteronyssinus	Class 4	Severe bronchial asthma	Step 4 - high dose ICS-LABA	1 in last 1 year	Urban
							Der. farinae	Class 4				
52)	Bhagyashree	9 years	F	Cough+cold on and off	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
53)	Chirag	10 years	M	Cough+cold and breathing difficulty	Cold weather	Allergic rhinitis	Der. pteronyssinus	Class 1	Mild persistent allergic rhinitis	Oral antihistamines+ intranasal corticosteroids	Nil in last 1 year	Urban
							Der. farinae	Class 2				
54)	Pratiksha	8 years	F	Cough+cold+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
55)	Dharshini	9 years	F	Cough+cold+noisy breathing	House dust	Bronchial Asthma+ Allergic rhinitis	Der. pteronyssinus	Class 4	Moderate bronchial asthma + mild persistent Allergic rhinitis	Step 3 - medium dose ICS-LABA	2 in last 1 year	Urban
							Der. farinae	Class 4				
56)	Bhoomika	6 years	F	Fever+running nose	House dust and pollen	Allergic rhinitis	Grass mix 2	Class 1	Mild persistent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
57)	Vijaykumar	12 years	M	Cough+cold and breathing difficulty	House dust and pollen	Bronchial Asthma	Grass mix 2	Class 4	Severe bronchial asthma	Step 4 - high dose ICS-LABA	3 in last 1 year	Urban
							Birch	Class 1				
							Mugwort	Class 2				
							Der. pteronyssinus	Class 1				
							Der. farinae	Class 2				
							Cat	Class 4				
							Aspergillus fumigatus	Class 1				
58)	Mohammed Iqlas	7 years	M	Cough+cold+running nose	House dust	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban

59)	Samarth	8 years	M	Cough+cold on and off	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Rural
60)	Sreenika	9 years	F	Cough+cold	House dust	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 1- Low dose ICS	Nil in last 1 year	Rural
61)	Rakshith	6 years	M	Cough+cold+noisy breathing	Not known	Bronchial Asthma	Nil	Nil	Mild bronchial asthma	Step 1- Low dose ICS	Nil in last 1 year	Urban
62)	Shreesha	7 years	F	Fever+running nose	Not known	Allergic rhinitis	Nil	Nil	Mild intermittent allergic rhinitis	Oral antihistamines	Nil in last 1 year	Urban
63)	Ananya	15 years	F	Cough+cold+noisy breathing	House dust and pollen	Bronchial Asthma	Der. pteronyssinus	Class 4	Severe bronchial asthma	Step 4 - high dose ICS-LABA	1 in last 1 year	Rural
							Der. farinae	Class 4				