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# Evaluation of the Profile and Treatment Results for Allergic Children

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## Abstract

**Background:** The majority of allergy sufferers produce IgE antibodies, which are antigen-specific antibodies; the term sensitivity addresses the clinical expression of IgE-mediated allergic disease.

**Methods:** This retrospective analytical study was carried out in the Pediatric Allergy and Immunology Unit, Muhammad Al-Mousawi Children's Hospital. The data included were those of 422 patients (286 males and 136 females). The mean age at disease onset was  $0.97 \pm 1.33$  years, and the mean duration of follow-up was  $4.31 \pm 2.93$  years. Data regarding bronchial asthma, course of the disease, treatment received and outcome at last visit were obtained from the patients' records. The ultimate objective was to evaluate the current epidemiologic and clinical profile as well as risk factors and the therapeutic responses of allergic diseases in our community.

**Results:** According to a statistical analysis of the patient records, allergic diseases were more prevalent in males than in females (ratio 2.1: 1) and in metropolitan than rural and rustic regions. Positive family background of other atopic sicknesses to be specific unfavorably susceptible rhinitis and atopic dermatitis was higher than positive family background of bronchial asthma in our series. The most common causes of bronchial asthma were environmental and viral factors. Dyspnea and cough were the most common symptoms of asthma exacerbation. Exclusive breastfeeding didn't appear to safeguard against the improvement of bronchial asthma. The prevalence of mild and moderate cases was higher than that of severe cases. Eosinophils and serum total IgE were both elevated in the laboratory, and the most common treatment in this investigation was inhaled corticosteroids.

**Conclusion:** The procedure can be used in other facilities and pertinent investigations, but these results are particular to our unit and should not be interpreted generally. The sample size and retrospective design of the study limit the validity of the findings and conclusions.



## Introduction

Over the past 30 years, the prevalence of common allergic diseases like asthma, food allergies, atopic dermatitis, and allergic rhinitis has increased. The search for treatment strategies that keep up with these mutations resulted in recent changes in the patterns of acquiring allergic diseases, which are increasing in both number and type [1]. The term 'sensitivity' refers to patients who express an 'altered state of reactivity' to common environmental antigens. The majority of allergy sufferers produce IgE antibodies, which are antigen-specific antibodies; the term sensitivity addresses the clinical expression of IgE-mediated allergic disease [1]. In natural conditions, allergens frequently function as proteolytic enzymes, which may enhance mucosal permeability and cause sensitization. Allergen is an antigen that triggers an IgE response in genetically predisposed individuals [2]. All humans are exposed to potential allergens. Genetically predisposed atopic people react by rapidly increasing their T helper 2 (Th2) cells, which produce cytokines that encourage the production of IgE and eosinophilia. Additionally, the proliferation of T helper type 1 (Th1) cells, which release cytokines like interferon (IFN), leading to the induction of allergen-specific immunoglobulin G (IgG) antibodies, is a response seen in non-atopic people. Because Th1 cytokines may activate phagocytes and encourage the production of opsonizing and complement-fixing antibodies, Th1 cells are frequently implicated in the destruction of intracellular pathogens like *mycobacteria* [3]. The Fundamentals of allergic dendritic cells, Langerhans cells, monocytes, and macrophages all play important roles in the development of allergic inflammation by exposing allergens to T cells and helping to recruit effector cells locally [4]. The ability to present antigens in the context of the major histocompatibility complex (MHC) is a characteristic shared by a diverse population of cells known as Antigen presenting cells. Dendritic cells and Langerhans cells, which are unique in their potential to activate naive T cells, mediate the primary immunological response, which is the sensitization stage of allergies [5]. Eosinophilia in the peripheral tissues and blood is a sign of allergic disorders. Eosinophils and intracellular dense granules, which are the origins of inflammatory proteins, such as the main essential protein, are among them. These inflammatory proteins include eosinophil-derived neurotoxin, peroxidase, and cationic protein [6]. Mast cell involve or trigger appropriate stimulation and are a variety of mediators that can have different effects on allergic inflammation and organ function. These include mediators associated with preformed granules (histamine, serine proteases, proteoglycans) and de novo synthesis and release of membrane-derived lipids,

cytokines, and chemokines. Cyclooxygenase and arachidonic acid lipoxygenase metabolites, which have strong proinflammatory activity, are the most significant lipid mediators produced by mast cells. Prostaglandin D2 is the primary cyclo-oxygenase and lipoxygenase product of mast cells, while the primary sulfidopeptide leukotrienes are Leukotriene C4 and their peptidolytic derivatives LTD4 and LTE4 [7]. IgE linked to the FcRI and a multivalent allergen often cross link to activate mast cells and basophils immune systems. Interleukin-4 and IgE enhance the expression of the cell surface receptor FcεRI on mast cells. Surface FcRI levels drop in patients getting treatment with anti-IgE antibodies that lower blood IgE levels, which may be of therapeutic interest [8].

## Methods

This retrospective study analyzed the medical records of 422 children with allergic diseases (286 males and 136 females). Of these, 197 (46.7%) were diagnosed with bronchial asthma, while 225 (53.3%) had other allergic conditions, including allergic rhinitis and food allergies. the instances that remained but were not covered by our research. Age on average at the start was 0.97 years, to 8.6 years.

### Outcome at last visit was categorized as:

Active enrolment state: enduring symptoms despite treatment adherence. Remission state is the absence of all asthma symptoms at the time of enrolment in participants who had not taken any asthma medication before the examination. Retrospective analysis of medical records was done, and the following information was gathered (where accessible) in an electronic database until analysis was completed:

### I-Clinico-epidemiological data:

Data collected included demographic and personal information (e.g., location, sex), age at disease onset, and duration of follow-up. Environmental variables, diets, some of the precipitating reasons include viral infections, endocrine variables, psychological problems, irritants including cold air and parental smoking, pungent scents, and drugs like aspirin and sulfonamide. Clinical signs include things such as sweating excessively, low grade fever, cyanosis, dyspnea, and coughing. Other atopic illnesses that can present in children include atopic dermatitis, allergic rhinitis, and food allergies. In the past, nursing or artificial feeding were the only options. Historically, weaning and feeding regimes. Outcome and disease development.

### II. Lab data currently available:

At the initial visit, a complete blood image is taken (Auto hematology analyzer mindray, BC 5000).

Westergren method At the initial visit, the erythrocyte sedimentation rate (ESR) was measured. At the initial visit, serum total IgE was determined using an ELISA kit Enzyme-linked immunoassay from the Elabscience firm.

### III. Images that can be obtained:

Chest X-ray taken plainly to check for infiltrations, hyperinflation, or effusions during the initial visit.

**IV. Data about the treatments:** Type, method, and compliance of the medication taken. Duration of therapy till enrollment.

### Analytical Statistics

First, straightforward calculations for frequency, mean, standard deviation, and range were performed. The students T-test for continuous variables and Pearson's Chi-square test for categorical data were then used to compare incidences of bronchial asthma in active and remission. All statistical analyses were performed using the Statistical Program for Social Science (SPSS) version 11.0 (SPSS Inc., Chicago, Illinois, USA), with a p-value < 0.05 considered statistically significant.

### Statistical Analysis

The Statistical Analysis System (SAS) tool, as of 2018, was used to identify the impact of various variables on research parameters. The t-test was used to determine the statistical significance of the differences between means. The chi-square test was used to assess the statistical significance of the comparison of percentages at the 0.05 and 0.01 levels of probability in this particular investigation.

## Results

The demographic, clinical, and laboratory data of the sample under study are shown in the tables and figures provided. All patients examined in this study included the illness associated with a specific form of asthma, with all underlying causes being enumerated below. Bronchial asthma emerged as the predominant allergic ailment among individuals diagnosed using the aforementioned methodology.

Table 1 displays the results of the static analysis, indicating a high level of significance ( $P \leq 0.01$ ) for the variables of sex and address. However, the variables of Age at onset and follow-up period are shown as the mean standard deviation. The most common symptoms during an exacerbation of bronchial asthma are documented in Table 3. The frequencies of these symptoms were found to be statistically significant. The statistical analysis revealed a substantial association between the feeding type and asthma incidence, as shown by the results presented in Table 4.

Demographic data	Allergic diseases (N=422)	P-value
Sex		
Male / female (ratio)	286 (67.77%)/ 136 (32.33%) (2.1: 1)	0.0001 **
Address		
Urban	288 (68.2%)	0.0001 **
Suburban	116 (27.5%)	
Rural	11 (2.6%)	
*Age at onset (years)	$0.97 \pm 1.33$	---
*Duration of follow-up (years)	$4.3 \pm 2.93$	---
** ( $P \leq 0.01$ ).		

**Table 1:** Demographic data of allergic patients.

Variable	Patients with bronchial asthma and other allergic diseases	
	Frequency	Percent
Family history of bronchial asthma	134	31.8%
Family history of other atopic diseases	38	9%
No related with family of history	250	59.2%
Total	422	100
P-value	---	0.0001 **
** ( $P \leq 0.01$ ).		

**Table 2:** History of family related with asthma

Symptoms	Cases of bronchial asthma (N=422)	
	Frequency	Percent
Dyspnea	209	49.5%
Cough	207	49.1%
Cyanosis	207	49.1%
Low grade fever	62	14.7%
Vomiting	37	8.8%
Profuse sweating	13	3.1%
P-value	---	0.0001 **
** ( $P \leq 0.01$ ).		

**Table 3:** Clinical symptom frequency in patients during acute bronchial asthma flare-ups.

Type of feeding	Bronchial asthma cases	
	Frequency	Percent
Absolute breast feeding	182	43.1%
Mixed or artificial feeding	28	6.6%
artificial feeding	212	50.3%
Total	422	100%
P-value	---	0.0001 **
** ( $P \leq 0.01$ ).		

**Table 4:** Type of infant feeding in the studied sample.

Of the 187 patients with available data, 133 (71.1%) had high IgE levels ( $\geq 100$  IU/ml), while 54 (28.9%) had normal IgE levels ( $< 100$  IU/ml). The prevalence of the condition was within the normal range, and no statistical data were presented for the other patients. However, it is worth noting that a substantial level of statistical significance was observed.

The illness outcome, regardless of whether it was active or stable, did not exhibit any variation based on the triggering cause of acute episodes. This discrepancy was seen to be statistically significant between the two groups, as indicated in Table 5.

A statistically significant difference in disease outcome (active vs. remission) was observed for patients with normal and low IgE levels, but not for those with high IgE levels.

Precipitating factors	Active state on enrollment	Remission state on enrollment	Total	P-value
Viral infection	15 (6.73%)	29 (14.57%)	44	
Cold air	25 (11.2%)	25 (12.56%)	50	1.00 NS
Indoor allergens	20 (8.97%)	24 (12.06%)	44	0.497 NS
Fumes	19 (8.52%)	24 (12.06%)	43	0.331 NS
Foods	17 (7.62%)	20 (10.05%)	37	0.578 NS
Puberty	21 (9.42%)	17 (8.54%)	38	0.402 NS
Psychological stress	6 (2.69%)	15 (7.54%)	21	0.037 *
Sulfonamides	0 (0.00%)	6 (2.69%)	6	0.049 *
Aspirin	7 (3.14%)	5 (2.51%)	12	0.794 NS
Without causes	93 (73.2%)	34 (17.09%)	127	0.0001 **
Total	223	199	422	---
P-value	0.0001 **	0.0001 **	----	---

\* (P≤0.05), \*\* (P≤0.01).

**Table 5:** Variation of the asthma outcome according to the precipitating factors which number 422 state.

## Discussion

The development and phenotypic manifestation of allergy illnesses are influenced by a variety of variables, including genetics, exposure to environmental allergens as well as general adjuvant factors such illnesses, cigarette smoke, and air pollution. The most important problem in the epidemiology of allergy diseases is identifying these causes. Exposure to allergens, exposure to adjuvant risk or protective factors, and pharmacological treatment are all examples of preventative measures.

Our work aligns with past research in this aspect. The presence of a familial allergy history or a personal history of asthma significantly impacts the likelihood of developing asthma in children. This observation has been substantiated by London *et al.*, who have shown that the strongest association with early-onset persistent asthma is seen in cases when parents had a history of asthma and allergy [9]. Separate research has shown that the presence of asthma and allergy illnesses within a family lineage significantly influences the likelihood of developing asthma [10]. The co-occurrence of allergic rhinitis (AR) and asthma has been officially referred to as combined allergic rhinitis and asthma syndrome (CARAS), aligning with the overarching notion of "one airway, one disease" that characterizes this disorder. Allergic rhinitis (AR) and asthma are distinguished by inflammation in the upper and lower airways, respectively, wherein the development of both conditions has common origins. Given that these phenomena are elicited by the same etiological agents, they exhibit concurrent manifestation, have a similar inflammatory cell profile, and are amenable to the same therapeutic interventions [11].

In a study conducted by Sun *et al.*, it was shown that individuals with tIgE levels below 610 kU/L and a sIgE/tIgE ratio over 15.0% exhibit the highest levels of sensitivity and specificity in predicting a favorable response to allergen immunotherapy [12]. The research conducted by Vidal *et al.*, showed that using allergen-

specific IgE levels greater than 9.74 kUA/L as a biomarker for assessing the effectiveness of allergen immunotherapy (AIT) resulted in a sensitivity value of 96.4% and a specificity of 100% [13]. Hence, in the case of individuals diagnosed with allergic asthma, the molecular attributes of IgE (including total IgE, specific IgE, sIgE/tIgE ratio, and the presence of polysensitization or monosensitization) as well as a familial predisposition to allergies may significantly contribute to the prognostication of the efficacy of allergen immunotherapy (AIT).

The masculine gender was a substantial risk factor for asthma and wheezing. This observation may reflect a sex-linked biological difference or may be due to different patterns of environmental exposure, as asthma development is influenced by the interaction between genetic and environmental factors. In a cross sectional concentrate by Remes *et al.*, included 344 children of farmers and 366 children of non-farmers between the ages of 6 and 13 year, the former group had fewer asthma and allergic diseases and were less likely to be sensitive to common allergens than the latter. However, the children of farmers (17.2%) and non-farmers (14.5%) had similar levels of sensitization to the other allergens [14]. The average age of onset in our study was one year table (1) Lam and Leung (2007) conducted a cross-sectional study on 942 asthmatic children in the asthma clinic of a Hong Kong regional hospital. The earliest onset of asthmatic symptoms occurred between the ages of 1 and 3 year, with a mean age of 3 years. More than 90% of the children had asthma symptoms before they were six years old [15]. Unfavorably susceptible illnesses as a rule group in a similar family, and this was obvious in our series. Family background of bronchial asthma was seen in 31.8% of family members of our example and family background of other atopic sicknesses was apparent in 9%. These findings contrast with those of Lam and Leung , who found that in their series, the family history of other allergic diseases was more prevalent than the family history of asthma [15]. In a recent study by Bjerg *et al.*, the commonness of asthma was 5.3% among youngsters 7 to 8 years of age. In kids without parental asthma or parental atopy, the commonness of asthma was 2.8% as it were [16]. There have been a plethora of studies looking into the possibility of links between environmental exposure and allergic disorders, but no conclusive findings have been reached. Because almost all of the studies were carried out in Western nations, it may not be appropriate to apply these findings to people living in other nations, including Iraqi. Tsai and Tsai conducted research in Taiwan to investigate the connection between diet and asthma symptoms and symptoms of the respiratory system in school-aged children. They discovered that



intake of sweetened drinks, eggs, and fruits was linked to a decreased risk of asthma symptoms whereas consumption of soy products and beverages and fruits was linked to a greater risk of respiratory symptoms and asthma symptoms [17]. Then again, Beausoeil *et al.*, came to the conclusion that avoiding certain foods or additives did not appear to alleviate asthma and that asthma alone was unusual as a symptom of a food allergy. 72% of stable cases were diagnosed with mild intermittent bronchial asthma, which is understandable considering the decreased likelihood of remission in chronic cases. Grading of bronchial asthma served as the only independent risk factor for active illness in the current research. A more extended follow up period would offer more solid data. A longitudinal prospective population-based cohort study may be the most effective study design for determining the natural course of asthma [18].

## Author Contributions

Conceptualization: Rana Ali Othman  
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 Formal Analysis : Thaer Ali Hussein  
 Funding Acquisition: Rana Ali Othman, Thaer Ali Hussein  
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## Competing Interests

The authors declare that they have no conflicts of interest.

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