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ORIGINAL A R T I C L E

Evaluation Role of IL-13 and Eosinophils in Adult Asthmatic Patients

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Abstract

Background Asthma is a chronic inflammatory disease primarily resulting from interactions between genetic and environmental factors, in which immunological mediators and cells play a crucial role. This study aimed to determine the role of IL-13 and eosinophilia in adult asthmatic patients.
Methods This case-control study was conducted on 69 patients (48 females, 21 males) with acute bronchial Asthma and their age range (18-70). Other 20 healthy subjects (11 females and 9 males) were included as a control group. The optical density of IL-13 in serum was detected by ELISA technique and from which IL-13 was evaluated according to standard carve. A complete blood count was performed for all blood samples to detect eosinophils number (cell/µl) by the RUBY system.
Results The results of the study showed that 31.9% of the patients were in the age group of (20-35) years, 27.5% of the patients had clinical manifestations in the age >20 years, and 69.6% of patients were females. Significant association of gender and age for cases and controls not demonstrated (p-value = 0.23 and 0.83 respectively). This study detected the distinct role of IL-13 and eosinophil in cases of Asthma compared to healthy controls (P value <0.001) and showed the gradual high of IL-13 in serum associated with an ordered increase in eosinophil count in peripheral blood.
Conclusion The results of the study showed that 31.9% of the patients were in the age group of (20-35) years, 27.5% of the patients had clinical manifestations in the age >20 years, and 69.6% of patients were females. Significant association of gender and age for cases and controls not demonstrated (p-value = 0.23 and 0.83 respectively). This study detected the distinct role of IL-13 and eosinophil in cases of Asthma compared to healthy controls (P value <0.001) and showed the gradual high of IL-13 in serum associated with an ordered increase in eosinophil count in peripheral blood.

Keywords: Asthma; IL-13; Eosinophil; ELISA

1 Introduction

Reversible airway obstruction is a sign of Asthma, a chronic inflammatory disease of the airways with no known cause. At least 5 to 10 percent of adults suffer from it, making it one of the most prevalent chronic conditions [1, 2]. Asthma can occur at any time in a person's life, even though the majority of cases begin

before the age of 25. Although Asthma appears to be a complicated process with multiple involved genes and likely gene-environment interactions, it has about 60% heritability, suggesting that both genetic and environmental factors play a role in its etiology [3].

Airborne allergens and viral infections appear to be the most significant environmental factors. In susceptible individuals, Asthma may also be brought on by diet, smoking, and air pollution [4, 5]. No newly discovered genetic variant has increased the risk for the asthma phenotype across all populations, despite this evidence of a substantial genetic contribution to the biology of Asthma and the identification of several candidate genes. According to studies, multiple genetic variants may be responsible for an individual's asthma heritability and may also play a role in the expression of the phenotype across a population. Genetic variants that influence treatment response have also been identified [5–8].

The various clinical manifestations that favor a primarily T helper type 2 (TH2) response, with interleukins such as interleukin-13 (IL-13) that lead to the formation of immunoglobulin E (IgE), proinflammatory cytokines, and bronchial hyperactivity, as well as the interaction between genetic and environmental factors (allergens), are the primary causes of asthma disposition [9]. Early asthmatic responses are triggered by T cells, derived cytokines, IgE, mast cells, and recruitment and activation of eosinophils, which appear to contribute to the persistent asthma phenotype with chronic airflow obstruction [10].

There is an increase in eosinophilopoiesis and subsequent migration of eosinophils to the lung in eosinophilic disorders like Asthma because: Type 2 cytokines (IL-4 and IL-13) upregulate chemokine production, including CCL11 (eotaxin 1), CCL24 (eotaxin 2), CCL26 (eotaxin 3), CCL13 (MCP4), and CCL5 (RANTES), which enhance chemotaxis for eosinophil trafficking from the circulation to the airway [11–13]. Elevated levels of IL-3, IL-5, and adhesion molecules like integrins on the surface of blood eosinophils are activated when these chemokines bind to the chemokine receptor CCR3. In turn, this makes it possible for eosinophils to interact with endothelial cells via periostin, intracellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1), which results in blood infiltration into the airway tissue [14]. Chemokine knockout mice like CCL11-/- and CCL24-/- show diminished eosinophil dealing to the aviation route during allergen challenge [15, 16]. In an Aspergillus fumigatus-actuated asthma model, CCR3 knockout mice had reduced eosinophilic aviation route aggravation alongside decreased degrees of type 2 cytokines, including IL-13 [17]. Our research aimed to determine the serum levels of IL-13 and eosinophils in patients with acute bronchial Asthma.

2 Materials and methods

2.1 Study design

The current research was case-control study conducted on 69 patients (48 males, 21 females) who were seen in Al-Diwaniya Teaching Hospital from December 2022 to January 2023. The patients were diagnosed clinically by a physician as having bronchial Asthma. Patients were interviewed directly by using an anonymous questionnaire that covered age, sex, duration of the disease, detailed history of occupation, smoking, the frequency of symptoms, any drug use, family history, and others. Another group consisting of 20 apparently healthy individuals (11 male and 9 female) without any history of systemic disease were clinically considered as healthy and also included in this study as a control group. This study was in agreement with the ethics of Al-Diwaniya Teaching Hospital, and verbal informed consent was obtained from all participants.

2.2 Sample collection

Two ml of blood in another anticoagulant tube are use immediately for a complete blood count. Three ml of blood in a sterile plain tube and allow the sample to clot for a few minutes at room temperature then, followed by separation of serum from the clot by centrifugation for 15 minutes at 1000 ×g. Then the serum was divided into two Eppendorff tubes, labeled, and stored at -70 °C for the IL-13 ELISA assay procedure.

2.3 Human IL-13 enzyme–linked immunosorbent assay kit

Cusabio (Germany) ELISA kits used for quantitatively determining IL-13 level in serum and assay procedure carried out according to the manufacture's manual.

2.4 Eosinophils count

Complete blood count was performed for each blood sample by the RUBY system. Assay procedure for this system involves taking an EDTA tube (containing blood samples and labeled with the name and number of the patient) in a specific rack in the RUBY system. Samples were taken and analyzed automatically by this system. After 1-5 minutes result of the complete blood count involving eosinophils count appeared on the computer screen. Finally result of each patient was printed and labeled with the name and number of the patient.

2.5 Statistical analysis

Data were translated into a computerized database structure. The database was examined for errors us-

ing range and logical data cleaning methods, and inconsistencies were remedied. Expert statistical advice was sought. Statistical analyses were done using SPSS version 20 computer software (Statistical Package for Social Sciences) in association with Microsoft Excel 2010.

3 Results and discussion

In this case-control study, 69 patients with acute bronchial Asthma were seen in AL-Diwaniya Teaching Hospital (21 males, 48 females), Table 1; the age of the patients varied from 18-70 years with a mean age of 34.4 years (SD±16), Table 2, compared with 20 (9 males, 11 females) healthy subjects with age range from 18 to 69 years, and mean age of 35.3 years (SD±17.3) as a control group. Table 3 showed most of the patients are females at a rate of 69.6%.

Table 1: Distribution of patients with bronchial Asthma over age groups and gender.

Age (years) Groups	Males No.	Females No.	Total No.(%)
<20	5	14	19 (27.5%)
20-35	6	16	22 (31.9%)
36-50	5	11	16 (23.2%)
>50	5	7	12 (17.4%)
Total	21	48	69(100%)

Table 2: The case-control difference in mean age.

Case-control comparison			
	Healthy controls	Cases (Asthma)	P
Age (years)			0.83[NS]
Range	(18 - 69)	(18 - 70)	
Mean	35.3	34.4	
SD	17.3	16	
SE	3.86	1.92	
N	20	69	

*S= No Significant, SD= Standard Deviation, SE= Standard Error, N= Number

Table 3: The case-control difference in mean age.

Case-control comparison					
	Healthy controls (n=20)		Cases (Asthma) (n=69)		
Gender	N.	%	N.	%	P
Female	11	55.0	48	69.6	0.23[NS]
Male	9	45.0	21	30.4	
Total	20	100.0	69	100.0	

The mean serum IL-13 and median eosinophil count were significantly higher among cases with asthma, 28.3 pg/ml and 979 cell/μL respectively, compared to healthy controls, 8.2 IU/ml, 4.8 pg/ml and 212 cell/μL, Table 4.

Table 4: The case-control difference in mean serum IgE, IL-13, and blood eosinophil count.

Serum IL13 conc. pg/ml	Case-control comparison		P Value
	Healthy controls	Cases (Asthma)	
Range	(3.3 - 7.7)	(13.8 - 48)	
Mean	4.8	28.3	
SD	1.2	9.1	<0.001
SE	0.27	1.1	
N	20	69	
Eosinophil count (cell/uL)			
Range	(61 - 351)	(671 - 3834)	
Median	212	979	<0.001
Inter-quartile range	(133 - 279)	(933 - 1279)	
N	20	69	

Table 5 shows the lowest range of IL-13 was 13.8 - 45.3 (mean ± SD=18.5 ± 7.2) associated with the lowest quartile of eosinophil (<933 cell/uL), the high range of IL-13 is 21.7-33.1(mean ± SD = 21.7±33.1) associated with increased number of eosinophil (interquartile range = 934 - 1279 cell/uL) and higher range of IL-13 is 32.2 – 48(mean ± SD = 32.2±48) associated with highest quartile of eosinophil (> 1280 cell/uL). A very strong and statistically significant positive (direct) linear correlation (trend) of IL-13 level with eosinophil count (r=0.917) also showed in these results.

Table 5: The eosinophil count by median serum IL-13 ordered categories.

	Eosinophil count (cell/uL)-quartiles			
	First (lowest) quartile (<= 933	Average-inter-quartile range (934 - 1279)	Fourth(highest) quartile (1280+)	P(ANOVA) trend
Serum IL13 conc. pg/ml				
Range	(13.8 - 45.3)	(21.7 - 33.1)	(32.2 - 48)	
Mean	18.5	27.8	39.7	
SD	7.2	3	5.6	<0.001
SE	1.7	0.51	1.36	
N	18	34	17	
r=0.917 P<0.001				

Table 6 demonstrated increase eosinophil number and high serum level of IL-4and IgE in asthmatic patients with positive family history (IL-4 mean ± SD=29.9±8, IgE mean ± SD=559.4 ± 179.7, eosinophil median =1152) whereas decreasing eosinophil count and low level of IL-13 in serum of asthmatic patients with negative family history with Asthma (IL-13 mean ± SD=25.2 ±10.5, eosinophil median =943).

Table 6: The mean and median of selected measurements by family history of Asthma.

	Family history of Asthma		
	Negative	Positive	P
Serum IL13conc. pg/ml			
Range	(13.8 - 47.3)	(13.8 - 48)	
Mean	25.2	38.9	0.04
SD	10.5	8	
SE	2.14	1.19	
N	24	45	
Eosinophil count (cell/uL)			
Range	(671 - 3834)	(740 - 2567)	
Median	943	1152	0.002
Inter-quartile range	(980 - 24)	(1298 - 45)	
N	24	45	

4 Discussion

Adult patients in our study with acute bronchial Asthma have the highest age and gender characteristics, with a prevalence of 31.9 percent among those aged 20 to 35 and 27.5 percent among those younger than 20. These differences may be attributable to high-life activities like work and exercise, which increase sensitivity to allergens, as well as hormonal changes in females (such as during pregnancy and menstruation), which may also increase asthma frequency [18].

These results concurred with after effects of Mishra (2004), who concentrated on 70 instances of bronchial Asthma at age 10-70 years, tracked down a high predominance of asthmatic patients during age 21-40 years, trailed by age 10-20 years and reduction with age [19]. Choi (2021) revealed that the abundance predominance of Asthma was most noteworthy in youngsters matured 6-17 years (15.3%), and it diminished with age for each more seasoned male and female; this is because of the connection between atopy and a raised eosinophil level in asthmatic cases was areas of strength for exceptional kids yet missing in the most established grown-ups class, until it was missing in grown-ups matured $\leq$ 55 years [20].

Mishra (2004) (19) found that the mean age of asthmatic patients (36.2 13.8 and 30.78 11.27 respectively) and controls (32.3 8.54 and 29.23 12.94 respectively) was P $>$ 0.05, and Vergara et al. (2010) [21] who studied 429 nonrelated adult asthmatics and 401 controls (mean age 36.15 18.32 and 34.98 17.8 years, respectively) found that this was consistent with our findings Li and others, In some populations (particularly urban ones), it was hypothesized that the prevalence of bronchial Asthma in older asthmatics might be the result of inadequate asthma treatment or healthcare, an increase in smoking or exposure to air pollution, and an increase in other pulmonary diseases or microbial infections in the airways [22].

The present study found that adult women had a higher prevalence of Asthma (69.6%) than men (30.4%). This suggests that adult females were particularly affected by Asthma because there is a greater chance that sex-related hormonal or biochemical differences may play a role in Asthma's pathophysiology. According to a different study, female sex hormones, which increase the secretion of IL-13 and total IgE, respectively, contribute to the higher prevalence of Asthma in adult women than in men. Conversely, progesterone prevents mast cells from releasing histamine, and estrogen stimulates FoxP3+ regulatory T (Treg) cells [23–25]. These results concurred with most investigations that viewed sex as a gamble variable of bronchial Asthma. According to the authors, the decreased clinical and immunological responsiveness directly related to hormonal changes accounts for the higher prevalence of Asthma in adult women than in men. Additionally, the effect of age on the prevalence of Asthma in each sex may be related to a difference in hormonal status, possibly influencing airway size, inflammatory conditions, and smooth muscle and vascular functions. However, Choi (2021) mentioned these differences because the airway caliber and lung function of adult males are greater than those of adult females smaller [26].

This result observed a significant association between the concentration of IL-13 and acute bronchial Asthma (P $<$0.001), possibly due to its essential role in the pathophysiology of acute bronchial Asthma. IL-13 plays an important role in eosinophil accumulation and is considered a critical factor in IgE synthesis by B cells, differentiation of naïve T-cells into Th2 effector cells, AHR, and airway inflammation [26].

According to the current study, asthmatic patients had a significantly higher level of cells in their peripheral blood (p=0.001). The central effector cell eosinophil, which is responsible for ongoing airway inflammation, may play a role in the pathology of Asthma by causing an increase in the number of these cells in Asthma. As a result, the cell may injure the airway mucosa and associated nerves [27, 28]

Through the release of lipid mediators, reactive oxygen species, and granule-associated essential proteins, which cause bronchoconstriction and mucus hypersecretion and damage nerves and epithelial cells [29]. Despite the well-documented association of tissue eosinophilia and eosinophil degranulation with several fibrotic syndromes and the fact that the cell is the source of several fibrogenic and growth factors, such as TGF-, TGF-, fibroblast growth factor-2, VEGF, matrix metalloproteinase -9, IL-13, IL-4, and IL-17, less attention was paid to the possibility that the eosinophil [30].

The effect of IL-4 on TH2 to produce IL-3, IL-5, IL-9, and GM-CSF, which stimulated bone marrow to the synthesis of new eosinophils, as well as IL-4's role in enhanced adhesiveness of the endothelium for eosinophils, was responsible for the gradual increase in eosinophil count in peripheral blood and the distinct role that IL-13 played in Asthma. Eosinophil cells likewise blend, store, and delivery IL-13 during the provocative reaction to rehash its capabilities (for instance, eosinophils collection) [31, 32].

Leukocytes derived from the bone marrow, eosinophils are uncommon in healthy individuals; however, type 2 cytokines—interleukins (IL)-4, -5, and -13—can speed up eosinophilopoiesis, extend eosinophil survival, and transport to the injury site during disease.

Eosinophilia, tissue damage, and airway pathology result from an abnormal inflammatory response in conditions like allergic Asthma. It has been demonstrated that the pleiotropic type 2 cytokine IL-13 plays a crucial role in developing Asthma and other eosinophilic conditions [33, 34]. IL-13 levels are raised in creature models of eosinophilic irritation and in the blood and tissue of patients with eosinophilic problems. Eosinophil survival, activation, and trafficking are just a few of the many pathogenic mechanisms that are triggered by IL-13 signaling. Information from preclinical models and clinical preliminaries of IL-13 inhibitors in patients have uncovered robotic experiences in the job of this cytokine in driving eosinophilia. Clinical trial results show that IL-13 plays an essential mechanistic role in Asthma and other eosinophilic disorders [32–35].

5 Conclusions

Our results showed that the concentration of IL-13 and the number of eosinophils increased in adults with bronchial Asthma compared with healthy subjects, and an increase in these immunological indicators was observed in patients with a positive family history. It also linked a positive linear relationship between IL-13 and the number of those cells.

Conflict of Interest: No conflicts of interest exist between the authors and the publication of this work.
Ethical consideration: The ethical committee approved the study at AL-Furat Al Awsat Technical University, Al-Diwaniyah, Iraq.

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