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THE CORRELATION BETWEEN SERUM TOTAL IGE AND IL-33 IN PATIENTS WITH ASTHMA AND ALLERGIC RHINITIS

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ABSTRACT

The present study was aimed to correlate the levels of total IgE with the levels of interleukin-33 in patients with asthma and allergic rhinitis.Total IgE was evaluate for 195 (109 males, 86 females) patients with airway allergic disease, 120 with allergic asthma (67 males, 53 females) their mean age was 31.16±1.73 and 75 with allergic rhinitis (45 males, 30 females) their mean age was 35.28±2.31. Serum IL-33 levels was astimated for 40 asthmatic (22 males, 18 females) and 35 allergic rhinitis patients (19 males, 16 females) compared with 15 healthy individuals controls, who attended the allergy specialized center in Baghdad /Al- Resafa from October 2014 to February 2015. A high significant ($P < 0.001$) increase in the level of total serum IgE in asthmatic patients (328.78 ± 33.07 IU/ml), and allergic rhinitis patients (379.76 ± 36.09 IU/ml) compared to healthy individuals (81.19 ± 8.04 IU/ml). In parallel IL-33 was also recorded a significant elevation in both groups of patients (128.07 ± 13.75 pg/ml) and (144.62 ± 12.78 pg/ml) respectively. While it was (59.04 ± 9.06 pg/ml) in healthy individuals. A significant positive correlation was found between serum level of IL-33 and the total IgE.

KEYWORDS: asthma, allergic rhinitis, total IgE, Interleukin-33.

INTRODUCTION

Allergy is an immune disorder occurs when the immune system react with non-infectious and normally innocuous environmental antigen[1]. Allergic diseases are considered the epidemics of the twentieth century which estimated to affect more than 30% of the population in industrialized countries with a still increasing incidence.[2] Asthma is usually connected to

allergic reaction or other forms of hypersensitivity.[3] Allergic rhinitis mainly caused by IgE-mediated reaction against inhaled allergens and involute mucosal inflammation driven by T-helper2 (Th2) cells.[4] Immunoglobulin-E (IgE) mediate the type I hypersensitivity reactions that contribute to the pathogenesis of allergic diseases such as asthma and allergic rhinitis.[5] Interleukin-33 (IL-33) is cytokine belong to the IL-1 superfamily and acts as important regulator in severe allergic disorders. The major purported mechanism of IL-33 in allergy is the activation of mast cell to produce a variety of pro-inflammatory cytokines and chemokines which play an important role in the pathogenesis of allergic rhinitis and asthma,[6,7] IL-33 binding to ST2 (IL33R) on a variety of cells such as mast cells and eosinophils, leads to mast cell proliferation and increased Interleukin-4 (IL-4) synthesis. This cytokine activate B cells to proliferate and produce IgE, which together with IL-33 and IL-4, stimulate mast cells to degranulate resulting in anaphylaxis.[8]

MATERIAL AND METHODS

The study was carried out at the specialized center of allergy in Baghdad /Al- Resafa from October 2014 to February 2015. The patients were classified into two groups, 120 with allergic asthma (67males, 53females) 75 with allergic rhinitis (45males, 30females) and 50 control. Blood samples were collected from all groups for estimation of serum total IgE by ELISA using a kit supplied by Dr.Foke (Germany), the value over 100 IU/ml were considered high and estimation of serum IL-33 by ELISA using a kit supplied by Cusabio (Chaines)

Statistical Analysis

Statistical analysis was performed to study the effect of different factors in study parameters using "The statistical Analysis System-SAS (2012) software". Least significant difference, LSD was used to significant comparison between means.

RESULTS

In this study a highly significant ($p<0.001$) increase in the serum levels of total IgE in asthmatic patients ($X= 328.78 \pm 33.07$ IU/ml) and patients with allergic rhinitis ($X= 379.76 \pm 36.09$ IU/ml) as compared with controls ($X= 81.19 \pm 8.04$ IU/ml) Table (1-1).

Table 1-1: The mean levels of T.IgE between studied groups.

Groups	T.IgE IU/ml			
	No.	Min.	Max.	Mean ±SE
Asthma	120	11.00	998.00	328.78±33.07*
Rhinitis	75	18.00	992.00	379.76±36.09*
Control	50	8.69	253.08	81.19 ±8.04

On the other hand IL-33 was significantly elevated in patients with asthma (X= 128.07 ± 13.75 pg/ml) and with allergic rhinitis (X= 144.62 ± 12.78 pg/ml), in comparison to controls (X= 59.04 ± 9.06 pg/ml) as shown in Table (1-2).

Table 1-2: Mean concentration of IL-33 in sera of studied groups.

Groups	IL-33 Pg/ml			
	No.	Min.	Max.	Mean ± SE
Asthma	40	25.30	257.40	128.07±13.75*
Rhinitis	35	29.40	236.90	144.62±12.78*
Control	15	15.20	126.20	59.04 ±9.06
*(P<0.01).				

Statistically, there was a positive correlation between total IgE levels and IL-33 in asthma and allergic rhinitis as shown in Table (1-3).

Table 1-3: Correlation coefficient between total IgE and IL-33 levels.

No.	Groups	Correlation coefficient (r)
1	**Asthma**	**0.42****
2	**Rhinitis**	**0.31***
****(P<0.01). *(P<0.05).**		

DISCUSSION

Asthma is a chronic inflammatory disease classically elevated serum IgE levels, and increased Th2 cytokine production.[9]

Allergic rhinitis is inflammatory disorder of the nasal mucosa, which is triggered by an allergen-IgE mast cell immediate reaction.[10] And this class of antibody plays an important role in the pathophysiology of these diseases.[11] Some studies have implicated the role of IL-33 in the pathogenesis of respiratory allergy and asthma.[12] IL-33 has been identified as a trigger of Th2 cell differentiation, which by interacting with both the innate and adaptive immune system can drive allergy and asthma pathogenesis.[13]

IL-33 is considered to play a crucial role in allergic inflammatory diseases including pathogenesis of asthma and allergic rhinitis.[14] The results of this study were in concordance with the results found by Guo *et al.* (2014),[15] who showed increased serum IL-33 levels in patients with asthma compared to those in non-allergic control and concluded that IL-33 is a marker of asthma severity, and may contribute to airway remodeling in asthma by acting on human lung fibroblasts. Other studies have also reported similar findings such as Azizy *et al.* (2014),[16] who approved that IL-33 and its receptor ST2 were markedly elevated in patients with bronchial asthma. This study showed agreement too with the results of Sakashita *et al.* (2008).[17] who reported significant increased serum level IL-33 in patients suffering from allergic rhinitis. On other hand, Asaka *et al.* (2012).[18] observed higher levels of IL-33 in nasal secretions from house dust mite sensitive patients with perennial allergic rhinitis. The elevated level of IL-33 was correlated with severity of allergic rhinitis symptoms, suggesting the role of this cytokine in pathophysiology of this disorder.[14]

CONCLUSIONS

This study confirms the noxious role of IL-33 in allergic diseases.

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