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EVALUATION OF SERUM INTERLEUKIN-17 AS DIAGNOSTIC MARKERS IN ASTHMA SEVERITY

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A B S T R A C T

Asthma continues to be a major health issue worldwide, with an increasing prevalence impacting over 300 million people and contributing significantly to morbidity and mortality. This study investigates the role of interleukin-17 (IL-17) in different asthma phenotypes, focusing on its potential as a biomarker for disease severity and control. The primary aim was to measure & compare the serum levels of IL-17 in asthmatic patients with healthy controls and to explore correlation of IL-17 levels with other inflammatory markers such as IgE, C-reactive protein (CRP), and eosinophil count. Additionally, the study sought to determine the relationship between IL-17 levels and asthma control status.

The study encompassed 140 participants, divided equally between asthmatic cases and healthy controls, showing IL-17 levels were elevated in asthma patients though not reaching statistical significance. A clear association was observed between elevated IL-17 levels and other biomarkers with deteriorating control of asthma symptoms.

While the direct correlation between serum IL-17 levels and asthma severity was not statistically significant, the study indicates the potential of IL-17 to be a contributing factor in severe asthma cases. Further studies are needed to confirm these findings and assess IL-17's utility in clinical practice for asthma diagnosis and treatment.

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1. INTRODUCTION

Globally, airway diseases are a primary cause of morbidity and mortality. The prevalence of asthma has significantly increased in recent years; it now affects more than 300 million people globally and accounts for 1000 fatalities per day (Global Asthma Network, 2018).

Whereas previously all patients had been treated as though there was a single pathological process that caused the disease, now there is recognition of multiple asthma phenotypes and the possibility that each may require a different course of treatment depending on the underlying pathology of the individual patient (Pavord et al., 2018). Different asthma phenotypes with various inflammatory patterns have been identified using non-hierarchical clustering of numerous clinical and biological parameters (Haldar et al., 2008). Various inflammatory subtypes of sputum cells can be identified through histological investigation, including eosinophilic, neutrophilic, mixed granulocytic, and paucigranulocytic (Simpson et al., 2006). Eosinophilic inflammation, which is caused by type 2 (T2) inflammation, is the most common category that has been

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studied. The cytokines interleukin (IL)-4, IL-5, and IL-13, which promote eosinophil development, maturation, maintenance, and recruitment to the airways, are expressed in T2 inflammation. For this phenotype, a variety of highly effective medications are available, including ICSs, oral corticosteroids, and cutting-edge biologic medicines that target specific cytokines. However, these medications are ineffective for persons with asthma who have other inflammatory phenotypes, particularly neutrophilic sputum (Pavord et al., 1999; Chung, 2016).

IL-17 cytokines, which cause epithelial cells to generate chemokines and cytokines that drive neutrophils to the site of the inflammation, are frequently linked to neutrophilic inflammation (Blauvelt & Chiricozzi, 2018). Psoriasis and ankylosing spondylitis are two rheumatological disorders where IL-17A is particularly relevant (Monin & Gaffen, 2018). Numerous studies have linked higher levels of IL-17A to more severe forms of asthma (Bullens et al., 2006; Al-Ramli et al., 2009).

Since the early 1990s, a prevalent paradigm has been that type 1 cytokine-secreting T-helper (Th1) cells are suppressed when T2 T-helper (Th2) cells are improperly activated, leading to atopic asthma (Godfrey et al., 2015). Other T-cell subsets and kinds of inflammation have, however, recently been recognized. Inflammation among these T-helper 17 (Th17) cells, characterized by the release of IL-17A, is thought to be essential in the body's defines against invasive bacteria and fungi (Monin & Gaffen, 2018).

A family of similar proteins is represented by the 155-amino acid secreted glycoprotein known as IL-17A in humans (McGeachy et al., 2019).

Genetic studies, clinical correlations, in vitro studies, and murine models have all shown a relationship between asthma and IL-17 dysregulation.

We will evaluate serum IL-17 as a diagnostic biomarker for asthma in this study and link it with the disease's severity.

The objectives of this study are to measure IL-17 levels in asthmatic patients and compare them with serum levels in healthy individuals; correlate IL-17 with IgE, Eosinophil count, and C-reactive protein (CRP); associate IL-17 with asthma control status; and calculate the cutoff point for serum IL-17 regarding asthma control status.

2. PATIENTS AND METHOD

This cross-sectional case-control research was carried out at Al-Imamain Al-Kadhimain Medical City / Baghdad from April 2021 to May 2022. One hundred forty participants were included in this study, of which 70 were cases of asthma and 70 were healthy controls. Control was matched by age and sex in the cases. The data on asthmatic patients were collected in the respiratory outpatient clinic.

Demographic data, and smoking history were obtained by direct patient interviews. Paper hospital records were used to obtain data on laboratory investigations. Blood samples were collected for the measurement of serum levels of C-reactive protein, IgE, complete blood count (Eosinophil count), and IL-17. Conventional Laboratory Protocols were used.

Inclusion criteria included known cases of asthma aged 18 and above were included in this research. Healthy controls are subjects without the disease being studied but may have other conditions indirectly affecting outcome.

Exclusion criteria were Patients with any concomitant pulmonary pathology, any type of malignancy and pregnancy women were excluded from the study.

CBC, C-reactive protein and IgE were analyzed at Al-Imamain Al-Kadhimain Medical City Educational Lab. Division using Automated system SIEMENS ATELLICA (CE approved). On the other hand, serum IL-17 were measured via the ELISA technique in the private sector under the supervision of a senior biochemist.

Reference normal laboratory values were as follows: C-reactive protein < 10 mg/L, IgE 150 – 300 (Sandeep et al., 2010) UI/ml, eosinophil count is less than 500 cells/mcL.

The Royal College of Physicians three questions (RCP3Q) was used to test for the control status of asthma (Pinnock et al., 2012), thus patients were divided into controlled, partly controlled, uncontrolled groups (Table 1).

Table 1. The Royal College of Physicians three questions for asthma

In the last month	**Yes**	**No**
Have you had difficulty sleeping because of asthma symptoms (including cough)?		
Have you had your usual asthma symptoms during the day (cough, wheeze, chest tightness or breathless)?		
Has your asthma interfered with your usual activities (e.g., housework, work, school, etc)?		

3. STATISTICAL ANALYSIS

Data was checked for normality using the Bar chart and Shapiro test, continuous variables were expressed as means and standard deviations or medians with interquartile ranges (IQRs) depending on whether the distribution of variables was normal or skewed.

Categorical variables were expressed as frequency and percentages. The Welch's t-test (for normally distributed variables) and Wilcoxon or Kruskal-Wallis ranks test (for non-normally distributed variables) were performed. The difference between categorical variables was investigated using either the $\chi 2$ test or Fisher's exact test, depending on the context. Spearman rho was performed to check the relationship between continuous variables. Univariate logistic regression analysis was conducted to assess the risk of uncontrolled asthma. The cutoff point for IL-17 in uncontrolled asthma was calculated used the Youden index method. A P-value less than 0.05 was considered statistically significant. R software was used for data processing, administration, and statistical analysis ("R version 4.1.3, R Foundation for Statistical Computing, Vienna, Austria").

4. RESULTS

Seventy cases and seventy controls were included in this research. the mean age for the cases was 45.9 ± 14.4 years. Males comprise 61% while females hold 39% of the cases. The control group was matched for age and sex to the case group. The median (IQR) of the blood biomarkers including IgE, CRP, Eosinophil count, and IL-17 was illustrated in Table 2.

When comparing cases and control, a statistically significant difference was found between the two groups regarding IgE, CRP, and Eosinophil count (P-value < 0.05). on the other hand.

IL-17 was higher in the cases with a median of 16 compared to 9 pg/ml, though the difference was not significant (P-value = 0.1).

Figure 1 show the distribution of asthma control status in the cases group.

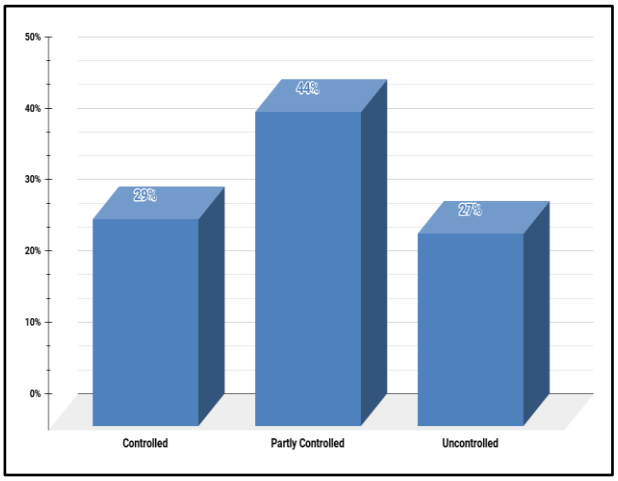

Figure 1. Distribution of Asthma control status in this study

Table 2. Baseline characteristics of patients and control and the statistical difference between the two groups

	Cases N = 70[1]	**Control N = 70[1]**	**P-value[2]**
Age, years	45.9 ± 14.4	45.1 ± 12.9	0.8
Male	43 (61%)	42 (60%)	0.9
Female	27 (39%)	28 (40%)	
Smoking	26 (37%)	27 (39%)	0.9
Eosinophil count, cells/mcL	276 (177 - 426)	97 (71 - 177)	**<0.001**
IgE, UI/ml	610 (439 - 824)	227 (175 - 295)	**<0.001**
C-reactive protein, mg/dl	1.4 (0.9 - 2.1)	0.9 (0.4 - 1.4)	**<0.001**
IL-17, pg/ml	18.6 (11.3 – 30.5)	11.2 (7.4-16.5)	0.1
[1]Mean ± SD; n (%); Median (IQR); [2]Wilcoxon rank sum test; Pearson's Chi-squared test			

Table 3 and Figure 2 summarizes the association between asthma control status and the blood biomarkers included in this study. A negative relationship was observed between the independent and the dependent variables, as the control status deteriorates the biomarkers rises. The blood level of IL-7, IgE, CRP and Eosinophil count was statistically associated with the control level in asthma patients (P-value <0.05).

Table 3. Association between asthma control status and the included blood biomarkers

	Controlled[1]	**Partly controlled[1]**	**Uncontrolled[1]**	**P-value[2]**
Eosinophil count	195 (165 - 301)	265 (176 - 387)	429.0 (351 - 486)	**<0.001**
C-reactive protein	1.2 (0.5 - 1.8)	1.2 (0.9 - 1.6)	2.3 (1.6 - 3.8)	**0.001**
IgE	411 (310 - 524)	643 (469 - 769)	943 (736 - 1,048)	**<0.001**
IL-17	16.3 (11.2 - 18.2)	19.2 (13.9 - 22.1)	25.2 (20.0 - 30.2)	**0.032**
[1]Median (IQR) [2]Kruskal-Wallis rank sum test				

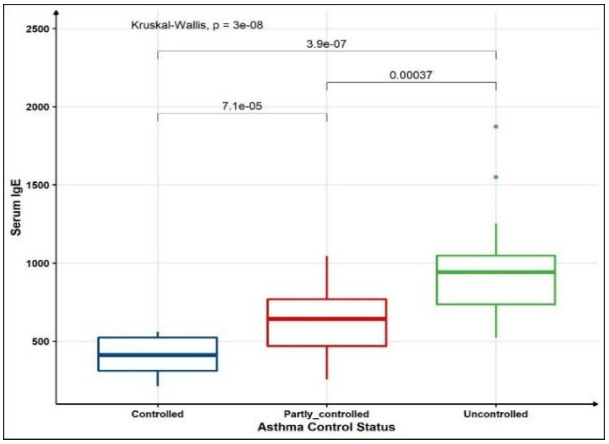

Figure 2. Boxplot showing the association between serum IgE and asthma control status

Correlation analysis using the Spearman correlation coefficient (rho) was conducted to study the relationship between IL-17 and other biomarkers. IL-17 was positively associated with IgE and the result was statically significant (rho = 0.24, P-value = 0.02) (Table 4).

A univariate logistic regression analysis (Table 5) found that high serum levels of Eosinophil count, C-reactive protein, IgE was associated with higher risk of uncontrolled asthma. Especially, high CRP level was associated with 70% more risk (OR = 1.7, P-value 0.007).

Table 4. Correlation analysis between IL-17 with IgE, Eosinophil count and C- reactive protein (CRP)

	IL-17	
	rho	**P-value[1]**
IgE	0.24	**0.02**
Eosinophil count	0.14	0.1
C-reactive protein	0.05	0.57
[1]Spearman's rank correlation, rho		

Table 5. Univariate logistic regression analysis of the association of IL-17, IgE, Eosinophil and the risk of uncontrolled asthma (severe form)

	OR	**95% CI**	**P-value**
Eosinophil count	1.2	1.01, 1.5	**<0.001**
C-reactive protein	1.7	1.20, 2.64	**0.007**
IgE	1.3	1.00, 1.72	**<0.001**
IL-17	1.05	1.01, 1.08	0.046
OR: odds ratio, CI: confidence interval.			

Using Youden index, cut-off point for IL-17 for different asthma control status was calculated (Table 6).

Table 6. IL-17 Cutoff point in different asthma control status

	Cutoff point	**Sensitivity**	**Specificity**	**AUC[1]**
IL-17				
Controlled	19.0	64%	99%	80%
Partly controlled	22.2	48%	56%	45%
Uncontrolled	28.6	84%	70%	86%
[1]Area under the curve; cutoff point calculated using Youden index method				

5. DISCUSSION

Our study showed that patients with asthma have markedly higher serum levels of IgE, eosinophil count, CRP, and IL-17 as compared to healthy controls which is demonstrated in Table 2. Though IL-17 serum level did not reach statistical significance in our study (P-value > 0.05), it was still higher in asthmatics compared to healthy controls. This is in line with what other authors demonstrated (Kubysheva et al., 2020; Dimitrova et al., 2019).

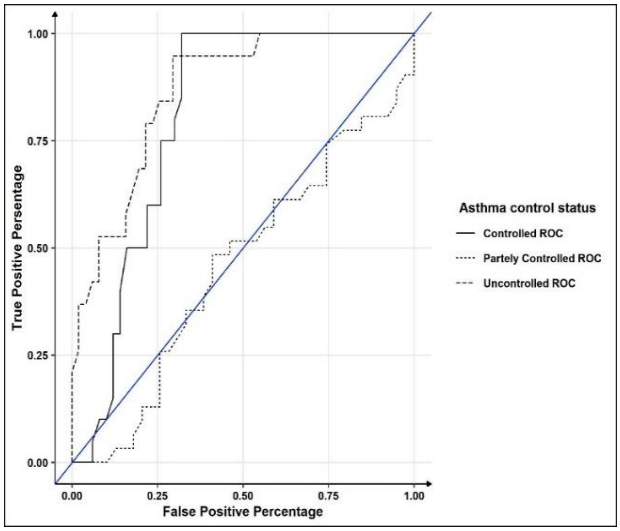

Figure 3. ROC curve showing the True positive and false positive percentage of IL-17 in different asthma control status

These were the only studies we found looking for an association of asthma with serum IL-17. Nevertheless, many other studies concluded a statistically significant association between asthma and IL-17 in asthmatic patients' sputum, bronchial lavage, or bronchial tissue samples instead (Moore et al., 2014; Ricciardolo et al., 2017; Bullens et al., 2006).

Such finding has been explained by the abundance of some IL-17-producing cell types in asthma patients like Th17, which was found to be higher in number in the peripheral blood of asthmatic patients compared to healthy people (Pridgeon et al., 2011).

Dimitrova et al, Tan et al., and Qian et al. confirmed our finding that asthmatics have higher serum levels of IgE and eosinophils (Dimitrova et al., 2019; Qian et al., 2008; Tan et al., 2018). Yavuz et al. came out with a similar conclusion studying asthma in children (Yavuz et al., 2021).

To study the reliability of using these parameters for evaluating and predicting asthma severity, we compared the serum levels of these biomarkers among the three asthma groups, which revealed a statistically significant inverse correlation (P-value < 0.05). Such that as the control status deteriorates, the biomarkers rise.

Agache et al. in their study evaluating the role of IL-17 in systemic inflammation of asthma, found a statistically significant difference in IL-17 serum levels in patients with severe as opposed to those with mild-moderate asthma (Agache et al., 2010), which comes in concordance with our results.

Although Kubysheva et al. stated that serum IL-17 values in patients with moderate and severe bronchial asthma were comparable (P-value > 0.05). Yet their study confirmed an inverse correlation between the serum

content of IL-17 and values of FEV1, FVC, and FEV1/FVC percentage (Kubysheva et al., 2020). Such finding may thus be used to positively relate serum IL-17 to increasing asthma severity even though the use of FEV1/FVC ratio to reflect asthma severity is still questionable (Kim et al., 2009).

Some authors studied the relation of IL-17 levels in sputum and tissue samples of asthmatic airways with asthma severity, and most of them confirmed that IL-17 in those samples was significantly higher in patients with the more severe in contrast to a milder degree of asthma and healthy individuals (Al-Ramli et al., 2009; Zhou et al., 2005; Chakir et al., 2003). The only study that deemed this correlation insignificant was that of Bullens et al. (2006).

One theory to support the claim that IL-17 is associated with more severe, treatment-resistant asthma is the steroid hypo-responsiveness in IL-17+ asthma found by many studies (Al-Ramli et al., 2009; Vazquez-Tello et al., 2013; Nanzer et al., 2013).

In support of our results, Qian et al. found an association between increased serum CRP and increasing asthma severity (Qian et al., 2008). On examining the potential association between asthma severity and serum IgE levels, Qian et al. concluded that such association does not exist (Qian et al., 2008). One possible explanation is what we mentioned earlier that different asthma phenotypes are related to varying biomarkers.

We found that an increase in serum level of CRP, IgE, and Eosinophil count was associated with a higher risk of having uncontrolled asthma, reflecting their promising role in predicting disease severity.

Nevertheless, this was not the case with IL-17 (OR 1.05, P-value 0.046). At the same time, Agache et al. concluded that values above 20 pg/ml are an independent risk factor for severe asthma (P-value = 0.000257) (Agache et al., 2010) . Considering that most of the patients in our case sample have IL-17 serum values of less than 20 pg/ml, it can be difficult to build an accurate comparison between the two results.

Another study by Östling et al. linked the presence of IL-17 in high levels in bronchial tissues to an increased incidence of asthma exacerbations (Östling et al., 2019), which may point to poor asthma control.

Based on the above, IL-17 may have a predictive value for worsening asthma control. However, such value is yet to be confirmed by further research focusing more on the measurement of IL-17 in serum samples, which is much easier and less invasive than induced sputum and bronchial tissue samples.

Qian et al. showed that those with higher serum CRP levels were at increased risk for severe asthma and that the higher the serum level, the more risk is (Qian et al., 2008). This is in line with our study that asthmatics with higher CRP have 70 % more risk of developing uncontrolled asthma than those with lower serum values. This may reflect a good predictive role of CRP for asthma severity.

Our study found that the association between IL-17 and blood eosinophil count was insignificant. This is in line with what Agache et al. found (Agache et al., 2010).

Nonetheless, Molet et al. demonstrated evidence that may present eosinophils as IL-17-producing cells in asthma patients (Molet et al., 2001) , suggesting a rational and possible explanation for such association to exist. Similar evidence was shown as well by Guerra et al. in a murine model (Guerra et al., 2017).

This disparity in results, however, can be explained by what some authors suggested that specific cellular and molecular markers can define different asthma phenotypes (Fahy, 2009), indicating that IL-17 may contribute more to the pathophysiology of certain phenotypes than others.

There were some limitations in our study. Firstly, lack of a longitudinal period to confirm the stability of severity patterns. Secondly, the causal relationship between the study biomarkers and asthma phenotypes was not investigated, as was the effect of some concurrent medical conditions or asthma treatment. Lastly, the small sample size in our study may limit the result generalizability.

In conclusion, IL-17 play a promising role in the diagnosis, treatment, and monitoring of asthma, along with evaluation of its severity. Besides, it demonstrated a predictive role for asthma outcomes in terms of control. However, the necessity to interpret their serum values in light of other confounding factors like age, race, treatment modality, asthma phenotypes, and presence of comorbid conditions is to be considered and investigated further to maximize their accuracy in the proposed roles. Since it is much easier and less invasive to obtain and investigate serum samples than sputum, bronchial lavage, or biopsy samples. Further studies focusing on evaluating IL-17 levels in the serum of asthma patients are recommended.

A study correlating the local (pulmonary) and systemic (serum) IL-17 levels is highly recommended and would make the comparison of current IL-17-related studies much more manageable.

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