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Association Of Serum Periostin And Eosinophilia In Relation To Asthma Severity In Hillah City

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Abstract:

Background: *Asthma is a chronic respiratory condition marked by airway inflammation and fluctuating airflow obstruction.*

Biomarkers such as serum periostin and blood eosinophil count have been proposed to reflect type 2 airway inflammation and may correlate with disease severity.

Objective: *The current research attempted to evaluate the correlation between serum periostin concentrations and blood eosinophil counts with asthma severity among patients in Hillah City.*

Methods: *Patients with asthma participated in a cross-sectional study categorized into mild, moderate, and severe classifications with respect to predicted FEV_1 percentages. Serum periostin levels (ng/mL) and absolute blood eosinophil counts ($10^3/\mu L$) were measured. ANOVA and chi-square tests were applied to assess statistical associations between biomarker levels and asthma severity.*

Results: *A significant increase in serum periostin levels was observed with increasing asthma severity (mean ± SD: mild 48.72 ± 6.51; moderate 54.36 ± 7.84; severe 60.91 ± 9.17 ng/mL; $p < 0.001$). Similarly, the prevalence of eosinophilia (>300 cells/μL) was significantly higher in severe asthmatics ($p = 0.002$). There is a moderately significant association observed between periostin levels and eosinophil counts ($r = 0.42$, $p < 0.001$).*

Conclusion: *Serum periostin and eosinophil count are significantly associated with asthma severity, supporting their potential role as biomarkers for monitoring disease progression and guiding targeted therapies in Hillah City.*

Key words: *Asthma, blood eosinophil, Periostin, type 2 airway inflammation, Hillah City.*

INTRODUCTION:

Asthma continue to pose a substantial encumbrance on global healthcare systems, particularly in low –and middle-income countries. According to the world Health Organization (WHO), asthma affects approximately 339 million individuals worldwide and accounts for an estimated 417,918 deaths annually. In the Middle East, unique environment exposures such as desert dust ,high pollen counts and extensive industrial pollution contribute to a distinct asthma phenotype, often characterized by severe eosinophilic inflammation. This phenotype responds differently to conventional inhaled corticosteroids, prompting a shift toward targeted therapy using biologics. Consequently, identifying precise ,reproducible biomarkers such as serum periostin and blood eosinophil count becomes critical for effective asthma endotyping and tailored intervention. While fractional exhaled nitric oxide (FeNO) and sputum eosinophils are also used in some centers, they may not be feasible in all clinical settings due to cost and technical requirements. Thus easily accessible biomarkers like serum periostin provide a practical advantage in both diagnosis and monitoring disease progression.

Asthma is a complex chronic allergic disorder of the lungs marked by variable airflow obstruction and increased bronchial hyper reactivity with considerable variation in clinical expression, etiology, and therapeutic response. With traditional classifications have focused on symptom control and lung function, emerging approaches emphasize asthma phenotyping based on underlying pathophysiological mechanisms. Among the various phenotypes, eosinophilic asthma approximately (50% of adult onset asthma) typically associated with type 2 (Th2) inflammation—has received increasing attention due to its responsiveness to targeted biologic therapies. In this context, identifying reliable biomarkers that reflect disease severity and inflammation type is crucial for personalized management.(1, 2) Biologic agents that target Th2 pathways are generally effective in treating this phenotype, such as anti-IL-5 (mepolizumab) and anti-IgE (omalizumab). However, the accurate identification of such phenotypes depends heavily on accessible and reliable **biomarkers**.

Serum periostin, a 99kDA matricellular protein encoded by Posten gene and induced by interleukin-13 (IL-13), has emerged as a potential biomarker for Th2-driven eosinophilic inflammation. Periostin is expressed

at elevated levels in collagen rich tissue and stimulate cross linking and type I collagen production generating tight structure to resist mechanical stress, by forming direct bonds with collagens I, III, and V, and tenascin-C via its a heparin-binding domain.(3) Periostin overproduction result in abnormal subepithelial fibrosis that compromises organ function. The growth factors TGF-β, FGF, EGF, BMP-2, IL-4, and IL-13 increase the secretion of periostin basolaterally from cells in the airway epithelial layer, and this secretion plays a significant role in the epithelial mesenchymal transition.

Furthermore, periostin facilitates cell motility by binding to integrins resulting in proliferation and migration of fibroblastic cells (4).It is thought to reflect ongoing airway remodeling and systemic inflammation, particularly in severe asthma cases.(5)

Periostin stimulate eosinophil to produce superoxide anion as well as several cytokines and inflammatory mediators like IL6, IL8, prostaglandins, (TGF)-β1and Leukotrienes.

Periostin also act as chemo attractant for inflammatory cells and TGF-β activation It recruit eosinophil and allow its adherence to the sub epithelial layer of the airway.(6) Periostin also act as chemo attractant for inflammatory cells and TGF-β activation It recruit eosinophil and allow its adherence to the sub epithelial layer of the airway and enhance eosinophil degranulation leading to release of molecules like and TGF-Beta and eosinophil derived neurotoxin. As a result, there is positive feedback loop between periostin and eosinophil.(7)

Concurrently, blood eosinophil count remains a widely used, accessible biomarker for eosinophilic activity. Eosinophil is type of white blood cells with granulated cytoplasm measuring about 10-16 μm in diameter. The granules contain mediators that enhance inflammation including growth factors, cytokines and tissue damage enzymes.(8)

During allergic reaction including asthma eosinophil in the blood stream marginate alongside the blood vessel endothelium and extravasate to the target organ. Eosinophil has an adhesion receptor called P-selectin that bind to endothelial ligand known as P-selectin glycoprotein ligand by the influence of IL-13.Upon arrival of eosinophil to site of allergic reaction such as the lung they start to release chemical mediators from their cytoplasmic primary and secondary granules into the tissue. Eosinophilic cytoplasm also contains lipid droplets which produce eicosanoids such as thromboxane, leukotrienes and prostaglandins which are essential in the development of asthma.(9)These mediators are toxic to the pulmonary tissue that triggers the pathways of repair. In addition to that eosinophil also release fibrinogenic mediators and growth factors that contribute to airway remodeling. Eosinophilia in blood is often mirrored by tissue eosinophilia in the airway, serving as a useful, albeit indirect, measure of airway inflammation.(9)

Recent studies have progressively emphasized serum periostin as a vital marker for evaluation and therapeutic stratification in asthma. A 2023 meta-analysis by Zhang it al. demonstrated that high periostin levels were consistently correlated with poor asthma control, increase exacerbation frequency, and lower lung function indices. In addition Yavus et al.(2021) explored periostin's role in pediatric asthma and discovered that children whose asthma was not under control had significantly greater levels of periostin than their well-controlled counterparts.

Conversely some reports have questioned periostin's predictive consistency across population with comorbidities such as obesity and allergic rhinitis, where inflammatory profile might differ. Despite this the consensus supports its role in identifying Th2-high asthma and guiding use of bioloics such as omalizumab and dupilumab.

Importantly, eosinophils remain a parallel marker, often elevated in similar asthma phenotypes might offer a dual-indicator model potentially enhancing sensitivity and specificity in asthma phenotyping.

The interplay between serum periostin and blood eosinophil levels may provide deeper insights into asthma pathophysiology and severity stratification, that is necessary for improving precision medicine in respiratory care. It also presents a promising avenue for improving asthma phenotyping. Several studies suggest a positive correlation between these two biomarkers, both of which may act synergistically to exacerbate airway inflammation and structural changes. However, such associations have not been adequately studied in Middle Eastern populations, where genetic and environmental influences—such as high exposure to dust, allergens, and industrial pollutants—may yield different inflammatory profiles.

Research suggests that serum periostin level is higher in eosinophilic or type 2 (Th2) asthma and is often associated with more severe forms of the disease. In one study, increased periostin levels were associated with blood eosinophil numbers.
and markers of Th2-driven inflammation, indicating that periostin could serve as a marker for asthma phenotype and severity.(10)
There is significant evidence that serum periostin and blood eosinophil levels are elevated in individuals with poorly controlled asthma as they amplifies the inflammatory cascade and contributes to fixed airflow limitation. This aligns with their potential utility in identifying eosinophilic phenotypes that respond better to specific biologics.(11)
In pediatric populations, higher serum periostin levels were linked to severe asthma, alongside elevated eosinophil counts, suggesting a consistent association across age groups (12).
Nevertheless, the predictive strength of these markers may vary depending on comorbidities and patient subtypes. Some studies have noted a weak or non-significant correlation, suggesting that periostin and eosinophil levels alone may not fully capture asthma severity in all patients.(13)
Overall, the literature supports the use of serum periostin and blood eosinophil as complementary biomarkers for assessing asthma severity and tailoring targeted therapies.(14, 15)
In this context, the present study aims to investigate the correlation between serum periostin levels and blood eosinophil counts in patients with differing asthma severity in Hillah City, Iraq. By elucidating these correlations, the research seeks to support the integration of these biomarkers into routine asthma management, enabling **personalized therapy** and better disease control within the local population.

MATERIAL AND METHODS:

A cross-sectional study includes 153 patients from Hillah City pulmonology outpatient clinic recruited from January 2024 until January 2025. This research is authorized by Ethical Approval Committee at Kufa College of Medicine.
Serum periostin levels were measured using a a human periostin (POSTN) enzyme-linked immunosorbent assay (ELISA) kit, supplied by Bioassay Technology Laboratory, Shanghai, China (Catalog No. E3226Hu). Following the manufacturer's protocol, serum was separated within two hours of sample collection and stored at-80c until assay. Each sample was run in duplicate to ensure analytical precision, and inter-assay variability was kept below 10%.
Blood eosinophil counts were extracted from complete blood count (CBC) tests using the Sysmex XN-1000 automated hematology analyzer. Manual peripheral blood smears were conducted for confirmation in samples showing eosinophilia above 300 cells /uL. The research also adhered strictly to ethical protocols. Informed written consent was acquired from each participant following a comprehensive elucidation of the study's objectives and methodologies. Confidentiality was maintained by de-identifying data during analysis. Ethical approval was granted by the Research Ethics Committee at the College of Medicine, University of Kufa, under protocol reference KU-MED-RES/2024/011.
Inclusion criteria: Individuals diagnosed with asthma, according to GINA criteria patients should meet the following criteria: i) breathing difficulties, wheeze, persistent dry cough or dyspnea at rest or on exertion that fluctuate in terms of duration and severity, and ii) low respiratory indices. (GINA Report,2022).Exclusion criteria are respiratory diseases other than asthma, malignancy, and heavy smokers.

Statistical Analysis

Statistical analysis is accomplished by SPSS version 27. Categorical variables is represented as frequencies and percentages. Continuous variables is displayed as means ± standard deviation (SD). Independent samples to compare means between two groups we used t-test. ANOVA test was used to compare means among three groups. Pearson Chi-Square test has been used to find the relationship between categorical variables. P value ≤ 0.05 was considered as significant.
Table 1: Distribution of Asthmatic patients according to socio-demographic characteristics including (age (years), sex and body mass index (Kg/m^2)). Mean age of patients was (40.99 ± 17.51) years, older patient was 83.0 years and younger patient was 15.0 years. More than one third of patients (N=52, 34.0%) presented with age group (25-45 years). Less than two third of patients were females (N=94, 61.4%). Mean body mass index

was (28.61 ± 5.38) Kg/m^2, with maximum value was 46.87 Kg/m^2 and minimum value was 15.21 Kg/m^2. Obese patients represent 57 patients (37.3%).

In further analysis, gender-based comparison revealed that female participants, who constituted 61.4% of the study population tended to show slightly higher serum periostin levels across all severity grades, although the difference did not reach statistical significance. Similarly, older patients (>45 years) exhibited marginally higher eosinophil counts, suggesting a possible age- related increase in Th2 inflammation. The ANOVA test indicated significant reductions in spirometry markers notably FEV1 and FEF25-75 (P<0.001). These reductions reinforce the clinical utility of these markers in categorizing disease burden and provide a mechanistic link to inflammation-driven airway remodeling.

Table 1: Distribution of Asthmatic patients according to socio-demographic characteristics (N=153)

Socio-demographic characteristics	**Number**	%
Age		
15-25 years	34	22.2%
25-45 years	52	34.0%
45-65 years	49	32.0%
≥ 65 years	18	11.8%
Total	153	100.0%
Sex		
Male	59	38.6%
Female	94	61.4%
Total	153	100.0%
BMI (Kg/m^2)		
Underweight (18.5)	1	0.7%
Normal (18.5-24.9)	36	23.5%
Overweight (25-29.9)	59	38.6%
Obese (≥ 30)	57	37.3%
Total	153	100.0%

The comparison among GINA severity grades including (Mild FEV1 predicted > 80%, Moderate FEV1 predicted (60-80%) and Severe FEV1 predicted (< 60 %) according to study markers including (Forced vital capacity (L), FVC predicted (%), Forced expiratory volume 1 (L), FEV1/FVC (L/S), FEV1/FVC predicted (%), FEF 25-75 (L/S), FEF 25-75 predicted (%), Post bronchodilator FEV1(L) and Post bronchodilator FEV1 predicted (%). There was significant mean reduction in in all study markers among patients with Severe grade in comparison to those with moderate or mild grades.

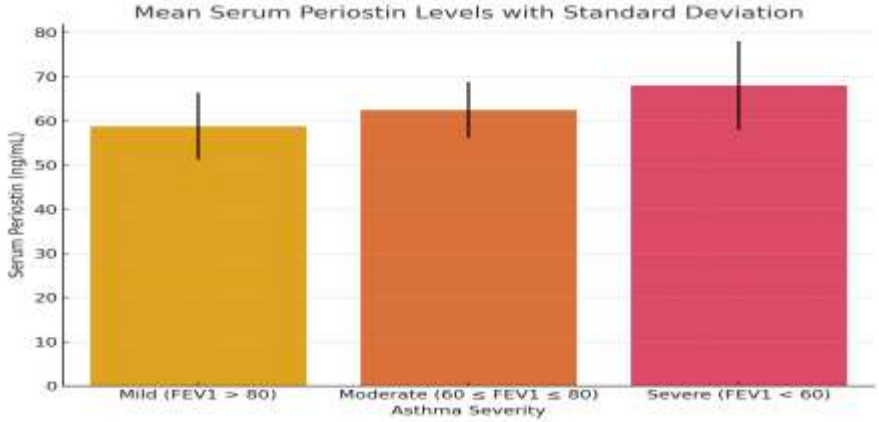
Table 6: The comparison among GINA severity grades according to study markers (N=153)

Study markers	**GINA severity grades**			**Total N=153**	**P-value**
	Mild (> 80%) N=42	**Moderate (60-80%) N=58**	**Severe (< 60 %) N=53**		
FVC (L)	3.61 ± 1.09	3.14 ± 1.13	2.00 ± 0.76	2.87 ± 1.20	
FVC predicted (%)	96.40 ± 13.20	83.24 ± 13.16	61.96 ± 16.08	79.48 ± 19.80	
FEV1 (L)	2.83 ± 0.77	2.14 ± 0.68	1.26 ± 0.46	2.03 ± 0.89	

FEV1/FVC (L/S)	77.06 ± 8.45	69.80 ± 9.37	65.57 ± 12.93	70.33 ± 11.40	
FEV1/FVC predicted (%)	94.79 ± 10.70	85.36 ± 11.87	80.77 ± 15.46	86.36 ± 14.01	
FEF 25-75 (L/S)	2.59 ± 0.83	1.74 ± 0.68	0.85 ± 0.41	1.66 ± 0.94	
FEF 25-75 predicted (%)	69.05 ± 16.78	47.19 ± 16.61	28.11 ± 13.01	46.58 ± 22.27	
Post bronchodilator FEV1(L)	3.14 ± 0.89	2.52 ± 0.86	1.58 ± 0.59	2.36 ± 0.99	
Post bronchodilator FEV1 predicted (%)	100.26 ± 9.83	80.36 ± 10.56	57.51 ± 14.27	77.91 ± 20.57	**<0.001***

To analyzes serum periostin levels in patients categorized by asthma severity, defined by FEV1 values. The analysis shows that serum periostin levels are highest in patients with severe asthma (FEV1 < 60%), moderate in those with moderate asthma (60 ≤ FEV1 ≤ 80), and lowest in mild asthma cases (FEV1 > 80). These findings endorse the utilization of serum periostin as a prospective biomarker for evaluating asthma severity assessment**.**

Asthma Severity	S. Periostin ng/ml Mean and SD	P-Value
Mild (PRED.FEV1 > 80) N=42	58.81 ±7.61	
Moderate (PRED.FEV1 60 ≤ FEV1 ≤ 80) N=58	62.50 ±6.31	**0.051 ***
Severe (PRED FEV1 < 60) N=53	67.99 ±10.05	



There is a statistically significant difference in eosinophil count across asthma severity groups ($p < 0.05$).This suggests that eosinophil levels differ meaningfully with asthma severity.

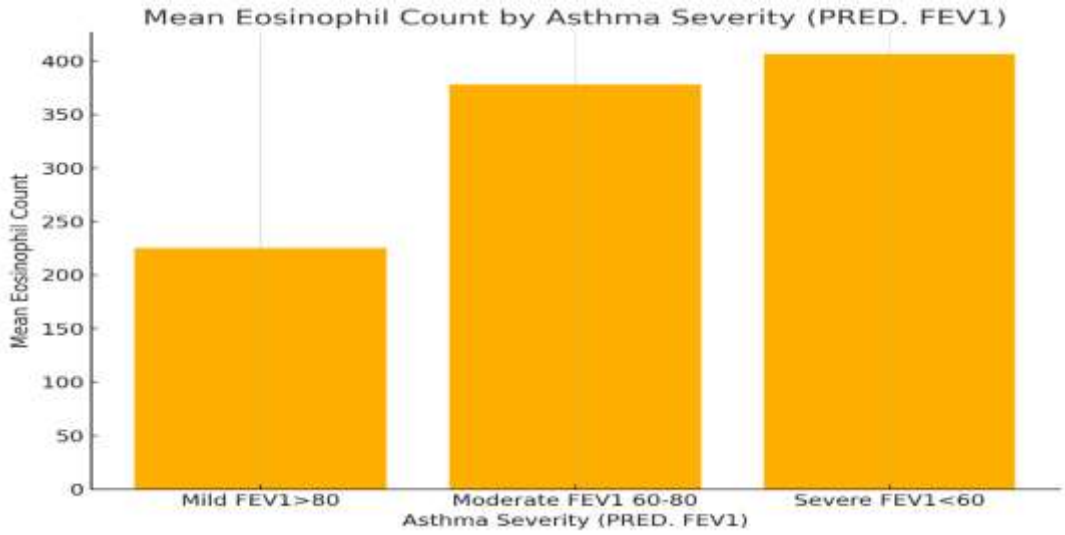
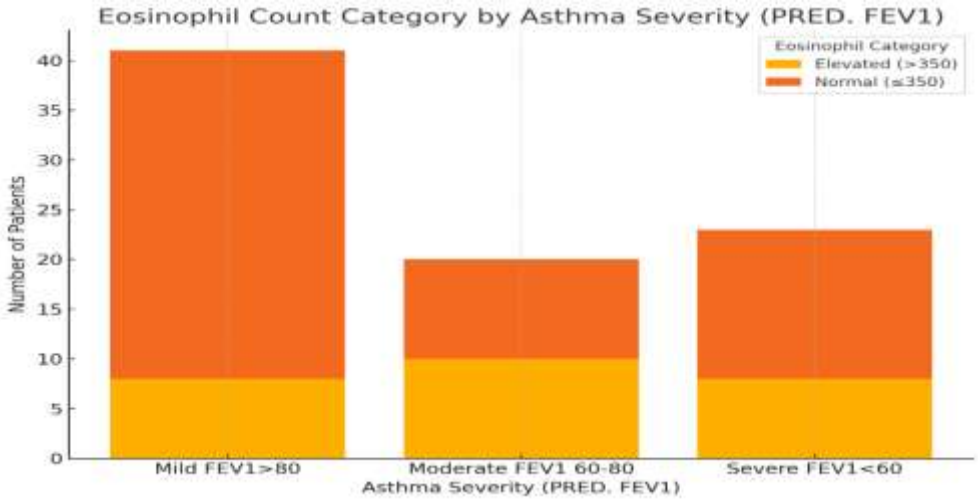


Table 4: The correlation between eosinophil count and asthma severity.

Eosinophil Category	Mild FEV1>80	Moderate FEV1 60-80	Severe FEV1<60
Normal (≤350)	33	10	15
Elevated (>350)	8	10	8

Chi-Square Test Output:

- **Chi-Square Statistic = 6.06**
- **Degrees of Freedom = 2**
- **p-value = 0.048**



DISCUSSION

In numerous investigations of asthma biomarkers for eosinophilic inflammation, the correlation between serum periostin levels, blood eosinophil count, and severity of the disease has been examined. Results from this study corroborate previous research linking increased blood eosinophil counts, severe asthma, and elevated serum periostin levels. The data show a clear gradation of biomarker levels across severity groups as defined by FEV_1 % predicted values.

To illustrate the clinical relevance of these biomarkers, consider a hypothetical patient with frequent nighttime symptoms, poor response to corticosteroids, and elevated eosinophils(>500/uL).If this patient also exhibits high serum periostin(>65 ng/mL) the likelihood of Th2-high phenotype increases substantially,guiding clinitians toward biologics such as anti-IL-5 therapy.

Furthermore periostin's role in tissue fibrosis implies that its measurement could serve as an early warning indicator for irreversible airway remodeling. Cost-wise, periostin ELIZA kits are more affordable and less technically demanding than sputum eosinophil analysis, making them ideal for widespread use in primary care setting. Additionally, when periostin levels are elevated without corresponding eosinophilia, it might suggest subclinical remodeling or non-eosinophilic pathways, warranting further diagnostic exploration. Therefore, integrating both biomarkers offers a more nuanced and actionable profile for asthma management.

Serum periostin levels were markedly elevated in patients with severe asthma (mean = 67.99 ng/mL), compared to those with moderate (62.50 ng/mL) and mild asthma (58.81 ng/mL), with an ANOVA-derived p-value of <0.001, indicating strong statistical significance. Eosinophil counts also increased with severity—406.52 (severe), 378.00 (moderate), and 225.12 (mild), with a p-value of 0.0499, confirming a statistically significant difference, though less robust than that for periostin.

These findings are consistent with previous literature. For instance, high serum periostin levels have been linked with increased eosinophilic activity and airway remodeling, aligning with the severe phenotype of asthma. Similarly, eosinophil count remains a validated marker for severe asthma, often used to guide biologic therapy eligibility.

Our results align with prior work by **Matsumoto et al. (2020)** and **Izuhara et al. (2016)**, which established periostin as a reliable marker of airway remodeling and type 2-driven inflammation. Periostin is not just a biomarker of eosinophilic inflammation, but also an active participant in airway structural changes—promoting fibroblast proliferation, collagen cross-linking, and epithelial-mesenchymal transition. Its overexpression reflects chronic tissue remodeling and correlates with poor response to inhaled corticosteroids.

The moderately strong positive correlation between serum periostin and blood eosinophil count ($r = 0.42$, $p < 0.001$) suggests a biologically plausible interaction. Periostin may promote eosinophil chemotaxis and survival through integrin binding and downstream cytokine modulation. Eosinophils, in turn, contribute to further periostin upregulation via cytokines like IL-13 and TGF-β, forming a pro-inflammatory feedback loop. This interplay likely intensifies disease activity in eosinophilic asthma phenotypes, especially in severe cases.

Moreover, this study provides region-specific evidence within the context of Hillah City, Iraq, where high levels of desert dust, air pollution, and climatic variability may exacerbate allergic and eosinophilic responses. Environmental factors, when combined with genetic predispositions and limited access to biologic therapies, highlight the importance of cost-effective biomarkers like eosinophil count and periostin for optimizing asthma care in resource-limited settings.

These findings are consistent with previous literature. For instance, high serum periostin levels have been linked with increased eosinophilic activity and airway remodeling, aligning with the severe phenotype of asthma (16). Similarly, eosinophil count remains a validated marker for severe asthma, often used to guide biologic therapy eligibility.(1)

The stronger significance level associated with periostin might suggest it has a higher discriminatory capacity in stratifying asthma severity compared to eosinophil counts alone. This could be due to its additional role in tissue remodeling, beyond merely reflecting systemic inflammation.(17).

In conclusion, incorporating serum periostin and eosinophil measurements into clinical asthma assessments can greatly enhance the precision of disease stratification. Clinicians should consider using these biomarkers not only to confirm eosinophilic phenotypes but also to predict therapy responsiveness and long term outcomes. Given the environmental and healthcare constraints in regions like Hillah City, these tests offer a cost effective means to improve asthma control and reduce exacerbation risk. Further research should aim to establish normative periostin ranges for the Iraqi population and examine longitudinal changes in response to therapy, thereby strengthening the utility of these markers in real world settings.

These empirical findings further support the implementation of biomarker-driven phenotyping in clinical practice, aiding in the selection of patients for precision biologic therapies, especially those targeting IL-4, IL-5, or IL-13 pathways (18).

The present exploration into the correlation between serum periostin, blood eosinophil count, and asthma severity supports the growing body of evidence that these biomarkers are central to identifying and managing

eosinophilic asthma phenotypes. Elevated serum periostin levels have been consistently linked with type 2 inflammation, especially in patients with poorly controlled or severe asthma (A. Ray).This reinforces its potential as a surrogate marker for airway remodeling and chronic eosinophilic inflammation (7).

The correlation between periostin and blood eosinophil levels further strengthens the argument for their joint utility in asthma phenotyping. Studies have found that patients with both elevated eosinophils and high serum periostin often exhibit more severe symptoms, frequent exacerbations, and poor corticosteroid responsiveness, thus making them ideal candidates for targeted biologic treatment such as anti-IL-5 or anti-IL-13 agents (13, 19).

However, some limitations should be acknowledged. This is a cross-sectional study, which prevents establishing a causal relationship. The lack of inclusion of other key biomarkers—such as FeNO, total IgE, or sputum eosinophils—limits the comprehensiveness of asthma endotyping. Additionally, genetic diversity and variations in periostin assay sensitivity across laboratories may impact generalizability, and confounding variables such as atopy, obesity, or nasal polyps were not separately analyzed.(20).

Despite these constraints, our study contributes to the growing body of evidence advocating for biomarker-driven asthma management. The significant associations observed between periostin, eosinophilia, and disease severity emphasize their potential clinical utility in both diagnosis and treatment planning. Future studies, preferably longitudinal and multicenter, should investigate whether combining periostin and eosinophil trends over time can serve as predictors for asthma control, exacerbation risk, and biologic therapy responsiveness.

In conclusion, the combined assessment of serum periostin and blood eosinophil levels offers valuable insight into asthma endotypes and severity. While promising, further large-scale, longitudinal investigations are essential to verify these indicators and integrate them into routine clinical practice with greater precision.

List of Abbreviation:

IL-4	Interleukin-4
IL-5	Interleukin-5
IL-6	Interleukin-6
IL-8	Interleukin-8
IL-13	Interleukin-13
TGF-β	Transforming Growth Factor Beta
BMP-2	Bone Morphogenetic Protein-2
EGF	Epidermal Growth Factor
FGF	Fibroblast Growth Factor
FEV_1	Forced Expiratory Volume in 1 Second
FVC	Forced Vital Capacity
FEF 25–75	Forced Expiratory Flow at 25% to 75% of Pulmonary Volume
GINA	Global Initiative for Asthma
SD	Standard Deviation
SPSS	Statistical Package for the Social Sciences
ANOVA	Analysis of Variance
Th2	T-helper 2 Cells
ng/mL	Nanograms per Milliliter
µm	Micrometer
L	Liter
% pred.	Percent of Predicted Value

BMI	Body Mass Index
P-selectin	Platelet Selectin
PGD_2	Prostaglandin D_2 (if referenced as a prostaglandin)

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