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Associations of Serum Periostin with Disease Severity and Control among Iraqi Asthmatic Patients

Hayder Yaseen ALI[1], Haider Abdulhameed ALQARAGHULI[2], Raid J. Mohammed AL-TIMIMI[1]

[1] Department of Chemistry and Biochemistry, Al-Nahrain University, College of Medicine, Baghdad, Iraq
[2] Department of Medicine, Al-Nahrain University, College of Medicine, Baghdad, Iraq

Corresponding Author: Hayder Yaseen Ali ✉ haydar.yaseen.ali@gmail.com

ABSTRACT

Objective: Periostin is a matricellular protein that has been associated with type 2-driven airway inflammation and asthma pathobiology. This study aimed to evaluate serum periostin levels in Iraqi adults with asthma compared with healthy controls, and additionally it examined the associations with disease severity and the Global Initiative for Asthma defined control status. Furthermore, the study assessed the diagnostic performance using the Receiver Operating Characteristic curve analysis for discriminating asthma cases.

Materials and Methods: A comparative cross-sectional study was conducted at Al-Imamain Al-Kadhimain Medical City and in the laboratories of the Department of Chemistry and Biochemistry at the College of Medicine, University of Al-Nahrain in Baghdad, Iraq. A total of 90 asthmatic patients and 90 healthy controls matched for age, sex, body mass index, and socioeconomic status were registered between April 2025 and January 2026. Serum periostin levels were measured using an Enzyme-linked immunosorbent assay (ELISA) and analyzed using nonparametric statistics.

Results: Periostin levels were significantly higher in asthmatics compared to controls, median 51.8 ng/ml (IQR: 44.9 - 57.9) vs. 32.9 ng/ml (IQR: 26.8 - 40.1), p <0.001. Periostin increased significantly with disease severity (p < 0.001) and asthma control worsening (p=0.006), peaking at uncontrolled severe disease. Eosinophilic patients had higher periostin levels in comparison to the non-eosinophilic phenotypes, median 52.96 ng/mL (IQR: 47.9 - 59.5) vs 47.64 ng/mL (IQR: 41.9 - 52.8); p=0.003. Receiver Operating Characteristic curve calculation yielded an area under the curve value of 0.904, and a 95% Confidence interval 0.851 to 0.943, achieving 92.2% sensitivity and 73.3% specificity at a >38.5 cut-off.

Conclusion: These findings show that periostin is associated with inflammatory status and significantly associated with poor asthma control and increased disease severity in adult Iraqi asthmatics, supporting its utility as a clinically applicable biomarker for T2-high asthma phenotyping and disease monitoring

Keywords: Periostin, disease severity, T2-high inflammation, eosinophilic asthma, Iraq

INTRODUCTION

Asthma is a heterogeneous chronic inflammatory disorder of the airways characterized by episodic wheeze, airflow limitation, mucus hypersecretion, and bronchial hyperresponsiveness (1-3). Although asthma is traditionally viewed as an allergic eosinophilic disease, it is now recognized as a spectrum of phenotypes and biological endotypes linked to distinct molecular pathways, treatment responses, and prognoses (4). Type-2 (T2)-high asthma represents the most investigated endotype and is driven by the activity of key cytokines, including interleukin-4 (IL-4), interleukin-5 (IL-5), and interleukin-13 (IL-13), which orchestrate Immunoglobulin E (IgE) synthesis, eosinophil maturation and survival, and recruitment of inflammatory cells into the airway mucosa (5,6). Among these cytokines, IL-13 has emerged as a dominant effector molecule, inducing structural alterations such as goblet cell metaplasia and subepithelial fibrosis (7). Periostin is a 90-kDa matricellular protein originally isolated from osteoblasts, where it was shown to promote cell adhesion and spreading of fibroblasts and epithelial cells (8,9). It is

ORCID Hayder Yaseen Ali / 0009-0002-3934-3011, Haider Abdulhameed Alqaraghuli / 0000-0002-1401-1304, Raid J. Mohammed Al-Timimi / 0009-0009-4796-0934

encoded by the periostin gene and secreted into the extracellular matrix by airway epithelial cells and fibroblasts in response to IL-4 and IL-13 stimulation (2,10,11). Periostin acts downstream of IL-13 signaling and contributes to hallmark pathological processes, including epithelial remodeling, collagen deposition, and eosinophil survival in the airway microenvironment (12). Periostin similarly stimulates Transforming Growth Factor-β signaling, which can further facilitate the deposition of extracellular matrix and airway remodeling (13). Accumulation of periostin in subepithelial regions has been shown to enhance eosinophil adhesion and migration (14), supporting its strong mechanistic link to T2-mediated airway inflammation (5,15). Serum periostin gained prominence following reports demonstrating its association with airway eosinophilia, decline in lung function, and response to targeted therapies such as anti-IL-13 biologics (16). Beyond serving as a systemic marker of T2-high inflammation, periostin has been implicated in fixed airflow limitation and airway remodeling, even in clinically stable or Inhaled Corticosteroids treated patients. Based on this dual relevance, periostin is a promising biomarker that serves as a bridge between chronic structural alterations and inflammation (5,16,17). Despite the extensive global research, there is still a scarcity of studies from the Middle East, particularly Iraq. Periostin levels were substantially elevated in asthmatic patients compared to healthy individuals in a previous Iraqi study, and there was an inverse relationship between periostin levels and Forced Expiratory Volume at one second (FEV1%) (7). Nevertheless, the relationship between periostin and GINA-defined asthma control and disease severity in Iraqi populations has not been thoroughly examined. Therefore, this study evaluates serum periostin levels in Iraqi asthmatic adults compared with healthy controls and further investigates periostin variation across severity classifications and control levels, providing insight into the potential utility of periostin as a locally relevant biomarker of T2-driven disease activity.

MATERIAL and METHODS

Study Design and Population

A comparative cross-sectional study was conducted at Al-Imamain Al-Kadhimain Medical City and in the laboratories of the Department of Chemistry and Biochemistry at the College of Medicine University of Al-Nahrain in Baghdad, from April 1, 2025, to December 1, 2025. Participants were recruited using a simple random sampling method during one-month period. The study included 180 participants, comprising 90 adults with clinically diagnosed asthma and 90 healthy controls without respiratory disease. Asthma patients were diagnosed and classified under the supervision of a specialist physician according to GINA 2025 guidelines (1). Based on these guidelines, asthma severity is assessed retrospectively depending on the level of treatment required to achieve symptom control. The current study cohort comprised patients classified into two severity categories: moderate and severe asthma. Patients with mild asthma were not included in this study due to recruitment challenges in our clinical setting. Furthermore, patients were categorized according to symptom control and treatment response into three groups: well-controlled, partially controlled, and uncontrolled. Additionally, to define the inflammatory phenotype, asthma patients were stratified into eosinophilic and non-eosinophilic groups based on a peripheral blood eosinophil count of $\geq 0.4 \times 10^3/\mu L$ (18-20).

Inclusion and Exclusion Criteria

Participants were adults aged 18-70 years with a physician confirmed diagnosis of asthma. Exclusion criteria included: age <18 years; mild asthma; other chronic respiratory diseases (e.g., chronic obstructive pulmonary disease or pulmonary fibrosis); pregnancy or lactation in women; continuous use of systemic corticosteroids for more than 30 days; currently receiving allergen immunotherapy or treatment with biologic agents (e.g., the anti-IL-13 monoclonal antibody lebrikizumab) within a defined period prior to enrollment, as these treatments can lead to reduced periostin expression (21,22); a history of advanced liver disease (e.g., cirrhosis, advanced fibrosis, cholestatic liver disease, or end-stage liver disease) or chronic kidney disease; advanced malignant tumors; active smokers; and hypothyroidism (23).

Sample Collection and Biomarker Measurement

Venous blood samples (5 mL) were collected under aseptic conditions. Blood samples were allowed to clot at room temperature and were then centrifuged at 3000 rpm for ten minutes. The serum was isolated and preserved at –80°C until analysis. Serum periostin concentrations were quantified using a validated enzyme-linked immunosorbent assay (ELISA) kit supplied by Bioassay Technology Laboratory (China), according to the manufacturer's instructions. The studied biomarkers were measured for all participants at a single time point

Ethical Considerations

The study protocol was approved by the Institutional Review Board of the College of Medicine, University of Al-Nahrain, on 10-03-2025, with the decision number 223/3/2. All participants provided verbal informed consent and were asked to complete a prepared questionnaire. The structured questionnaire included patient ID, full address, phone number, age, sex, asthma triggers, and Asthma Control Test scores (24), which reflect the level of asthma control, smoking/environmental exposure, presence of chronic diseases, current medications, and selected clinical manifestations. Individuals were informed of their ability to withdraw at any time without any negative impact on their clinical care, and confidentiality was maintained throughout the study. The ethical standards outlined in the Declaration of Helsinki were adhered to in all procedures.

Statistical Analysis

Statistical analysis were performed using JASP (version 0.95.4) and MedCalc (version 23.4.0) for data processing, tabulation, and inferential testing, whereas GraphPad Prism (version 10.6.0) was used for graphical presentation. Continuous variables are presented as mean ± SD or median with interquartile range (IQR, 25th-75th percentile), as appropriate. All comparisons between two independent groups were performed with Student's t-test (if normally distributed variable) or the Mann-Whitney U test (if not normally distributed variable). The Chi-square test was performed for categorical variables. To compare multiple groups, the Kruskal-Wallis test followed using Dunn's post-hoc multiple comparison test was employed. The Receiver operating characteristic (ROC) curve was used to calculate the area under the curve and the cutoff value, and to determine sensitivity and specificity. Multivariate logistic regression was used to evaluate the independent association between serum periostin and asthma severity after adjustment for key confounders. A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

The present study reports a case-control analytical evaluation of 180 participants, comprising 90 asthmatic patients and 90 healthy controls. The asthmatic cohort included both moderate and severe disease phenotypes; 56 (62.2%) were classified as having moderate disease severity, and 34 (37.8%) were classified as having severe disease severity. Based on asthma control, 31 (34.4%) were controlled, 34 (37.8%) were partially controlled, and 25 (27.8%) were uncontrolled, according to the GINA 2025 criteria. The percentage distribution of asthmatic patients according to disease severity and level of asthma control is shown in Figure 1.

The baseline characteristics of the population studied are briefly outlined in Table I. There were no significant differences between patients and controls regarding gender (χ^2=0.023, p=0.879). Likewise, no significant age difference was observed between cohorts (p=0.265). Body mass index was also similar in the two groups (p=0.242), with the majority of individuals in both cohorts being classified as overweight or obese.

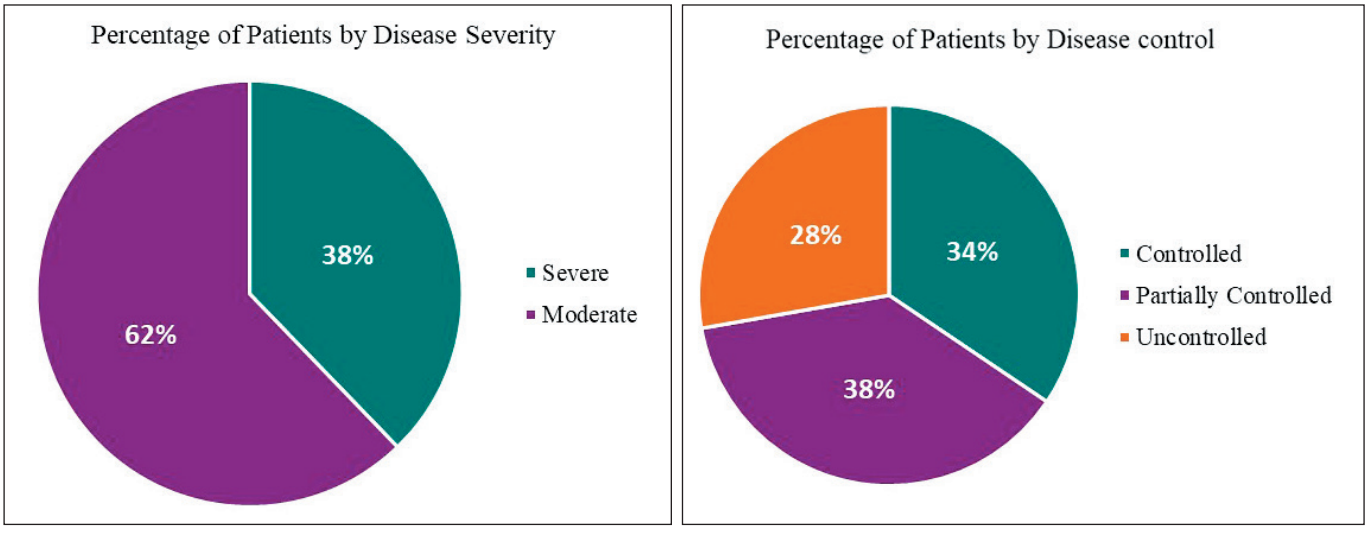

Figure 1: Pie charts showing the percentage distribution of asthmatic patients according to disease severity and level of asthma control

Table I: Baseline demographic and anthropometric characteristics of the study population according to gender, age, and Body Mass Index.

Parameter	Control (n=90)	Patient (n=90)	Statistical Analysis
Gender			
Male (n, %)	35 (38.9)	37 (41.1)	p=0.879
Female (n, %)	55 (61.1)	53 (58.9)	χ^2=0.023
Age (years)			
Mean ± SD	48.30 ± 12.69	46.00 ± 14.87	p=0.265
SEM	1.338	1.567	t=1.116, df=173.7
BMI (kg/m^2)			
Mean ± SD	29.36 ± 3.43	30.16 ± 5.55	p=0.242
SEM	0.362	0.585	t=1.173, df=148.4

Data are presented as frequency (n), percentage (%), and Mean ± SD. Comparisons of categorical variables were performed using the Chi-square test. Comparisons of continuous variables between two independent groups were performed using Student's t-test.
SEM: Standard Error of Mean. **BMI:** Body Mass Index.

Table II: Serum periostin concentrations in asthmatic patients and healthy controls according to disease severity, GINA-defined control status, inflammatory phenotype, and treatment variables

Parameter	Median	IQR	Statistical Analysis
Patients and Healthy Controls			
Patient (n=90)	51.80	(44.9 - 57.9)	**p < 0.001**
Control (n=90)	32.87	(26.8 - 40.1)	
According to the asthma severity			
Severe (n=34)	58.7	(54.7 - 68.6)	**p < 0.0001**
Moderate (n=56)	47.9	(43.5 - 52.4)	
According to the asthma control status			
Controlled (n=31)	45.9	(42.6 - 54.1)	
Partially Controlled (n=34)	52.6	(48.9 - 57.7)	**p=0.0060**
Uncontrolled (n=25)	53.8	(47.9 - 75.1)	
Eosinophilic and non-eosinophilic asthma			
Eosinophilic (n=55)	52.96	(47.9 - 59.5)	**p= 0.003**
Non-eosinophilic (n=35)	47.65	(41.9 - 52.8)	
Inhaled Corticosteroid (ICS)			
Yes (n=10)	52.23	(48.4-53.9)	**p= 0.974**
No (n=80)	51.51	(44.1-58.4)	
ICS / LABA Combination			
Yes (n=68)	50.87	(44.1-57.8)	**p= 0.377**
No (n=22)	52.44	(47.7-59.6)	

Data are presented as median and (IQR 25th-75th percentile). Comparisons between two groups were performed using the Mann-Whitney U test. Comparisons among multiple groups were performed using the Kruskal-Wallis test followed by Dunn's post-hoc multiple comparison test. LABA=Long-acting beta2-agonist

Table II demonstrates that serum periostin levels were significantly higher in asthmatic patients than in healthy controls, with median values of 51.8 ng/mL (IQR: 44.9 - 57.9) in the patients, compared with 32.9 ng/mL (IQR: 26.8 - 40.1) in the controls ($p < 0.001$). When stratified by disease severity, patients with severe asthma exhibited markedly higher periostin concentrations than those with moderate disease, with medians of 58.7 ng/mL (IQR: 54.7 - 68.6) versus 47.9 ng/mL (IQR: 43.5 - 52.4), ($p < 0.0001$). According to asthma control status, periostin levels increased progressively from controlled to partially controlled and uncontrolled groups. Median values rose from 45.9 ng/mL (IQR: 42.6 - 54.1) in controlled patients to 52.6 ng/mL (IQR: 48.9 - 57.7) in partially controlled and 53.8 ng/mL (IQR: 47.9 - 75.1) in uncontrolled patients. These differences were statistically significant across groups (p=0.006). Post-hoc Dunn analysis revealed two significant pairwise differences: (Partially Controlled > Controlled) and (Uncontrolled > Controlled), while no significant difference was detected between Uncontrolled=Partially Controlled, suggesting that periostin elevation begins early with loss of full control and remains comparably high in both partially and fully uncontrolled states. Periostin also showed a pronounced and statistically significant increase in eosinophilic asthma compared with non-eosinophilic patients. Median values were 52.96 ng/mL (IQR: 47.9 - 59.5) vs. 47.64 ng/mL (IQR: 41.9 - 52.8); p =0.003. These numerical trends are visually supported by the accompanying Table II and Figure 2.

No significant differences in serum periostin levels were observed according to inhaled corticosteroid (ICS) use (p=0.974) or ICS/LABA combination therapy (p=0.377). Furthermore, correlation analysis revealed that serum periostin levels were independent of demographic factors, showing no significant correlation with age (r=-0.033, *p*=0.756) or BMI (r=-0.081, *p*=0.448) while ROC analysis demonstrated strong diagnostic performance for periostin, which exhibited excellent discriminative ability in identifying asthma cases, with an AUC of 0.904, and 95% Confidence interval 0.851 to 0.943. At the optimal cut-off value (>38.5), periostin achieved high sensitivity

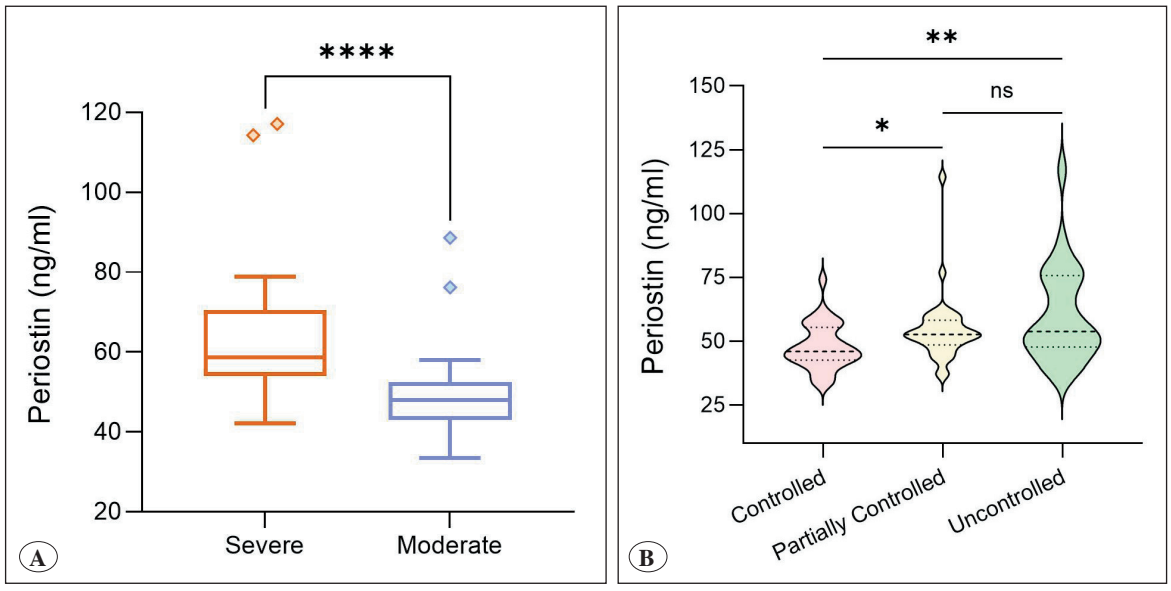

Figure 2: Serum periostin levels according to asthma severity and control status. **A)** Box-and-whisker plot with individual data points showing serum periostin concentrations (ng/mL) in patients with severe and moderate asthma. The central line represents the median, and the box indicates the interquartile range (IQR; Q1-Q3), and whiskers extend to values within 1.5×IQR. Points beyond this range are shown as outliers. Statistical comparison between groups was performed using the Mann-Whitney U test ($p < 0.0001$). Sample sizes: severe (n=34), moderate (n=56). Statistical comparisons between groups are indicated by brackets. **B)** Violin plot illustrating the distribution and density of serum periostin levels across GINA-defined asthma control categories (controlled, partially controlled, and uncontrolled). The internal lines represent the median and interquartile range (IQR). Statistical comparisons were performed using the Kruskal-Wallis test followed by Dunn's post hoc test (p=0.0060). Sample sizes: controlled (n=31), partially controlled (n=34), uncontrolled (n=25).

*Significance codes: *=$p < 0.05$, **=$p < 0.01$, ***=$p < 0.001$, ****=$p < 0.0001$, ns=not significant.*

(92.2%), while maintaining a moderately high specificity of 73.3% (Figure 3).

Multivariable logistic regression analysis was performed to evaluate the independent association of serum periostin with asthma severity after adjusting for potential confounders, including eosinophil count, age, and BMI. The overall model was statistically significant ($p < 0.0001$) with a Nagelkerke R^2 of 0.615. Serum periostin remained independently associated with severe asthma (OR=1.146, 95% CI: 1.065-1.233, p=0.0003). Eosinophil count was also a significant predictor (OR=56.27, 95% CI: 5.43-583.30, p=0.0007), whereas age and BMI were not significantly associated with asthma severity (Table III).

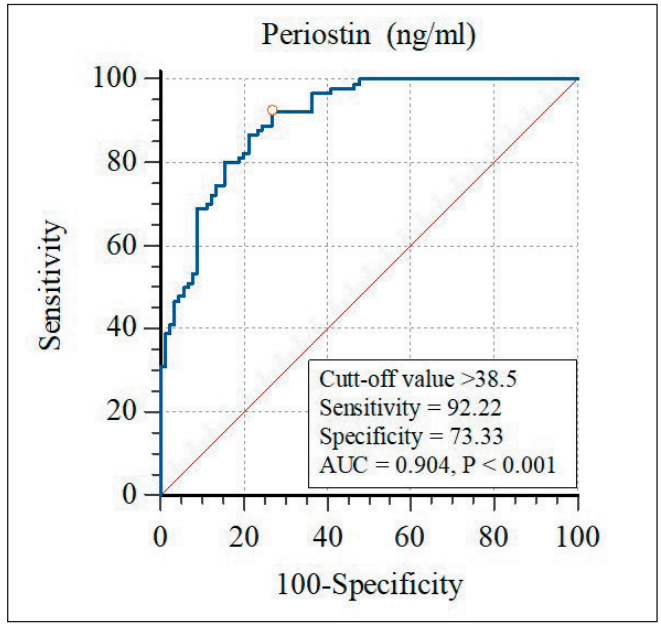

Figure 3: Receiver operating characteristic curve for (periostin) in classifying patients from controls.

DISCUSSION

The results of this study suggest that serum periostin is increased in Iraqi adult patients with asthma compared to control subjects, highlighting a strong association between elevated periostin levels and type 2 airway

Table III: Multivariable logistic regression analysis for predictors of asthma severity

Variable	Odds Ratio (OR)	95% CI	p value
Periostin (ng/mL)	1.146	1.065-1.233	0.0003
Eosinophils 10^3/μL	56.27	5.43-583.30	0.0007
Age (year)	1.048	0.999-1.099	0.052
BMI kg/m^2	0.883	0.774-1.008	0.065

Multivariable logistic regression analysis was performed to identify independent predictors of asthma severity (severe vs. moderate). Variables entered into the model included serum periostin, eosinophil count, age, and body mass index (BMI). Odds ratios (ORs) with 95% confidence intervals (CIs) are presented. A p-value < 0.05 was considered statistically significant.

inflammation. The significant difference in expression between patient samples and controls supports previous findings of marked periostin upregulation in asthmatic cohorts (7,25). More recent clinical studies have consistently reported elevated serum periostin levels in asthma compared to controls. For instance, Kim et al. demonstrated significantly higher serum periostin levels in patients with asthma compared to healthy controls, associating periostin with airway remodeling and hyperresponsiveness (26). Similarly, Ravindran et al. showed that periostin, when integrated with clinical and inflammatory profiles, contributes to distinct asthma phenotyping and clearly differentiates asthmatic patients from control groups (27). Earlier foundational studies have also demonstrated that periostin reflects eosinophilic airway inflammation and is closely linked to Th2-driven immune responses, further supporting the biological plausibility of the present findings (28). Periostin progressively increased across both clinical severity and GINA control categories, indicating that circulating periostin reflects the intensity and persistence of lower airway inflammation (29,30). Importantly, the association between periostin and asthma severity remained significant after adjustment for eosinophil count and other clinical variables, supporting its independent role as a biomarker of disease severity. This mirrors published observations that periostin tracks with airflow obstruction, tissue remodeling, and fixed airway limitation features, especially prevalent in moderate-to-severe asthma (31). The pattern seen in the current study, where poorly controlled individuals exhibited the highest periostin levels, suggests that systemic periostin may sensitively capture "residual" inflammation even when patients are under therapy. This finding is supported mechanistically by data showing that IL-13 directly induces periostin secretion from bronchial epithelial cells and may enhance eosinophil adhesion and trafficking within the extracellular matrix (5). Similarly, Baldo et al. reported periostin elevation in T2-high asthma with meaningful diagnostic contribution alongside Fractional Exhaled Nitric Oxide and IgE, highlighting periostin's complementary role in inflammatory phenotyping (4). Importantly, periostin varied independently of age or BMI in this cohort, suggesting that it reflects disease biology rather than demographic variation. This observation is relevant given recent evidence that obesity may distort other T2 biomarkers, reducing their reliability in overweight populations (31). In parallel, the associations of serum periostin with both GINA-defined loss of control and severity requiring higher treatment intensity underscore its potential role as an integrative biomarker reflecting persistent Type-2-driven airway pathology rather than symptoms alone. The higher levels of periostin among eosinophilic patients further reinforce periostin's value as a discriminative biomarker for T2-high asthma. Our observation is also consistent with the literature, placing periostin in line with T2-high biomarkers that frequently reflect eosinophil-associated inflammation, albeit with recognized modest relationships and assay/phenotype heterogeneity (32,33). The diagnostic effectiveness of periostin was further supported by ROC curve analysis, which demonstrated excellent discriminative ability (AUC=0.904). At the optimal cut-off value (>38.5), periostin showed high sensitivity (92.2%) and acceptable specificity (73.3%). These findings are in agreement with previous studies that evaluated periostin using ROC analysis. For example, Inoue et al. reported moderate diagnostic accuracy (AUC ≈ 0.70-0.75), while meta-analytic data suggest an overall AUC of approximately 0.87 for periostin in distinguishing asthma patients from controls (34). This was in line with other works indicating that periostin is a diagnostic marker to differentiate patients and controls (3,27). Compared to these reports, the higher AUC observed in the present study indicates superior diagnostic performance, which may be attributed to differences in patient selection, disease phenotyping, or population characteristics. Although systemic corticosteroid therapy is known to modulate T2-high inflammation and reduce periostin expression, we observed no significant differences in serum periostin levels in relation to ICS or ICS/LABA use in the present study. This finding is consistent with previous literature demonstrating that serum periostin levels may not significantly differ between asthmatic patients on or off inhaled corticosteroids (35),

as periostin often reflects refractory Th2/eosinophilic inflammation that persists despite standard inhaled therapies (36). Consequently, this suggests a limited treatment-related confounding effect in our cohort.

CONCLUSION

Serum periostin is significantly elevated in Iraqi adults with asthma compared to controls and is associated with disease presence. Higher periostin levels were also observed in relation to increased disease severity, poor GINA-defined control, and an eosinophilic phenotype, supporting its role as a clinically relevant biomarker reflecting type 2-driven airway inflammation. Periostin may contribute to improved diagnostic evaluation, disease monitoring, and phenotype stratification. However, further studies are warranted to confirm its discriminative performance and clinical utility across diverse populations.

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During the preparation of this manuscript, the authors used Gemini (Version 1.5 Pro, Google, USA) and Grammarly (Version 2024, Grammarly Inc., USA) solely to enhance the English language, grammar, and overall readability of the text. These tools were applied for linguistic refinement and editorial purposes only, without any involvement in data analysis, result interpretation, or the generation of original scientific content. The authors assume full responsibility for the integrity and accuracy of the final manuscript and affirm that these AI tools do not qualify for authorship.

Conflict of Interest

The authors declare no conflict of interest.

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Author Contributions

Concept: **HYA, HAA, RJMAT,** Design: **HYA, HAA, RJMAT,** Data collection or processing: **HYA, HAA,** Analysis or Interpretation: **HYA,** Literature search: **HYA, RJMAT,** Writing: **HYA,** Approval: **HYA, HAA, RJMAT.**

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