Serum Anti-Elastin Antibodies and Carotid Intima-Media Thickness as Indicators for Vascular Affection in Asthmatic Children

Original Article

Eman M. Fouda[1], Mariz zakaria Adly[2], Basma Sherif Fahmy[3], Asaad Gamal Asaad[4] and Heba Moustafa Hamza[1]

[1]Department of Pediatric Pulmonology, [2]Children's Hospital, [3]Department of Clinical Pathology, [4]Department of Diagnostic and Interventional Radiology and Molecular Imaging, Ain Shams University, Cairo, Egypt

ABSTRACT

Background: Asthma is associated with elevated cardiovascular disease risk, probably due to a common inflammatory pathophysiology.

Objective: This study assessed serum anti-elastin antibodies and carotid intima-media thickness (CIMT) as systemic vascular changes in asthmatic children and its correlation with asthma severity and control.

Patients and Methods: 70 children were enrolled in this prospective case-control study (35 asthmatic and 35 controls) from the Pediatric Pulmonology Unit, Children's Hospital, Ain Shams University, Cairo. They were subjected to a detailed history taking, physical examination, the following investigations were done (CBC, CRP, serum IgE, serum Antielastin antibody as a marker for vascular inflammation, carotid intima-media thickness by doppler ultrasound).

Results: Serum anti-elastin antibody levels were significantly higher in asthmatic group (69.11 ± 63.18 pg/ml) compared to control group (6.61 ± 3.68 pg/ml) (*P-value* < 0.001), with significantly higher level of serum anti-elastin antibodies in uncontrolled asthmatic children (91.16 ± 102.58 pg/ml) compared to controlled cases (51.05 ± 44.82 pg/ml) and partially controlled cases (68.85 ± 53.88 pg/ml) (*p-value* <0.001). A statistically significant positive correlation was discovered between serum IgE and anti-elastin antibodies (r= 0.774, *p*<0.001).

Carotid intima-media thickness (CIMT) among asthmatic children was (right 0.050 ± 0.008 cm, left 0.051 ± 0.007 cm) compared to control group (right 0.048 ± 0.007 cm, left 0.048 ± 0.009 cm) with percentage of increase 4% in right carotid artery and 5.4% in left one. No significant difference in mean CIMT between the right and left sides among asthmatic children with different asthma control levels.

Conclusion: Serum Anti-elastin antibodies a vascular inflammatory marker, were higher in asthmatic children compared to controls. This may require periodic follow up of any vascular changes in those patients.

Key Words: Asthmatic children, Antielastin antibody, carotid intima-media thickness.

Received: 11 February 2025, **Accepted:** 18 March 2025.

Corresponding Author: Mariz zakaria Adly, Resident, Children's Hospital, Faculty of Medicine, Ain Shams University, Cairo, Egypt, **Tel.:** +201098925592, **E-mail**: Mariz_zakaria@hotmail.com

ISSN: 2735-3540, Vol. 76, No. 2, June 2025.

INTRODUCTION

Asthma is optimally regarded as an immunologically mediated condition, where an abnormal immune reaction to diverse inhaled environmental stimuli and irritants causes a variety of events resulting in mucus hypersecretion, airway constriction, hyper-responsiveness, and eventually, symptomatic manifestations[1].

Elastin is a major component of the extracellular matrix. Changes in its metabolism are involved in the pathophysiology of destructive lesions of elastin-rich organs, such as blood vessels, kidneys, skin and lungs. Elevated levels of serum elastin-derived peptides (EDPs) are observed in emphysema, abdominal aortic aneurysm and atherosclerosis[2].

In asthma, increased proliferation of elastic fibers in the airways contributes to hyperresponsiveness and residual obstruction in asthmatic airways. Exacerbation of asthma may contribute to the production of elastin because hypoxia increases elastin secretion from arterial smooth muscle cells[3].

DOI: 10.21608/ASMJ.2025.356833.1389

Ultrasonography of carotid intima-media thickness can act as an available and dependable technique for identifying subclinical atherosclerosis. CIMT is markedly elevated in cases with already present plaques, serves as an indicator of subclinical organ damage, and functions as an independent predictor of cardiovascular and cerebrovascular diseases. Several studies have reported that associations exist between CIMT and established risk factors. Thus far, the current concepts of the risk factors of CIMT in the existing literature are not unified[4].

In adult cases, the progression of atherosclerosis follows the advancement of inflammation in asthma. This association has not been previously demonstrated in asthmatic children[5].

AIM OF THE WORK

The aim of this study was to evaluate serum anti-elastin antibodies and carotid intima-media thickness as systemic vascular changes in asthmatic children and its relation to level of asthma severity and control.

PATIENTS AND METHODS

Study Population:

This prospective case-control study was conducted at Pulmonology unit of Childrens' Hospital, Ain Shams University, from December 2023 till May 2024.

This study included 35 Children diagnosed with bronchial asthma according to GINA guidelines 2023 aged between 4 and 16 years old, and 35 healthy children with age and sex matched with patients.

Sample Size calculation:

After examining previous research results, the sample size has been determined utilizing power analysis and Sample Size Software (PASS 15) (Version 15.0.10), with a confidence level of eighty percent and a margin of error of ± 0.05. According to "Tokita et al", the expected mean serum anti-elastin antibodies among cases and controls is 67.4±29.7 ug/ml and 38.6±10.4 ug/ml respectively, and regarding "***Tarttersall et al.*** (2018)" the expected mean carotid artery IMT among cases and controls is 597±65 um and 558±42 um respectively. Depending on that, a sample size of 35 cases and 35 controls is going to be sufficient to attain research aim.

Study Tools:

All cases underwent a detailed history taking including demographic data, history of respiratory symptoms, age of onset, number of exacerbations in the last year, medication history, family history of bronchial asthma, other allergies for example atopic dermatitis and allergic rhinitis, exposure to allergens.

Physical examination including vital data, anthropometric measurements, complete chest examination, assessment of level of control and severity were done using GINA guidelines 2023[6].

The following investigations were done: CBC, C-reactive protein, total serum IgE, serum anti-elastin antibodies, carotid intima media thickness by ultrasound.

Serum anti-elastin antibodies:

Technique: Sandwich Enzyme-Linked Immunosorbent Assay (ELISA).

Specimen: Serum samples were permitted to coagulate for ten to twenty minutes at ambient temperature, followed by centrifugation at 2000-3000 RPM for twenty minutes to obtain the supernatant, then freeze at -20 ℃ till analysis. Specimens were completely thawed, homogenized, thoroughly vortexed then immediately assayed according to the manufacturer instructions (Chongqing Biospes Co., Ltd, China), absorbance was read on a spectrophotometer at 450 nm optical density (OD).

Calculation of results: Optical density of the samples was plotted on the standard curve according to the manufacturer instructions.

Carotid intima-media thickness:

All sonographic investigations have been conducted blindly by the same examiner. Images were obtained via high-resolution doppler ultrasonography (Samsung HS40) utilizing a probe LA3-16AD (3-16 MHz High Frequency linear transduce) conducted in radiology unit at Ain shams university. Three readings were taken in the same setting and the average was calculated.

Common carotid artery (CCA) intima-media thickness was measured in both carotid arteries. The average was taken of the measurements 1 cm above and below the largest measured place for the CCA intima-media thickness[7].

ETHICAL CONSIDERATIONS

The research was approved by the ethics committee of the Department of Pediatrics at Ain Shams University. Informed consent was obtained from the cases and their caregivers. Ethical Committee Approval Number: FMASU M S 634/2023.

Statistical Analysis:

The gathered data was coded, organized, and subjected to statistical analysis utilizing IBM SPSS Statistics software version 22.0, IBM Corp., Chicago, United States of America, 2013, and Microsoft Office Excel 2007.

Descriptive statistics were conducted for quantitative data, reporting the minimum and maximum of the range, in addition to the mean ± standard deviation (SD) for normally distributed quantitative data. For qualitative data, statistics were presented as numbers and percentages.

Inferential analyses for quantitative variables were conducted utilizing the Shapiro-Wilk test for normality and the independent t-test for two independent groups with normally distributed data. Inferential analyses for independent parameters in qualitative data were conducted utilizing the Chi-square test for differences in proportions and Fisher's Exact test for variables with small expected frequencies. The significance level was set at a *P value* of less than 0.050, indicating significance; alternatively, it is considered insignificant.

RESULTS

Our study included 70 children fulfilling the exclusion and inclusion criteria. They had been split into two groups, asthmatic and healthy children.

The present research discovered that no significant differences in age, gender, birth order, consanguinity, and residency were found between the groups (*p-value*= 0.135, 0.051, 0.592, 0.445, 0.320) respectively.

According to level of asthma control, 20% (N=7) were controlled, 62.9% (N=22) were partially controlled and 17.1% (N=6) were uncontrolled. As regards age of onset, both uncontrolled cases (1.93 ± 2.21 years) and partially controlled cases (2.08 ± 1.53 years) had earlier age of onset compared to controlled cases (4.78 ± 3.76 years).

Table 1: Comparison among asthmatic and control as regards demographic data.

		Group A Asthmatic (Number=35)		Group B Control (Number=35)		Test value	*P-value*
		N	%	N	%		
Age (year)	Mean ±SD	11.14 ± 3.08		8.10 ± 2.94		t=1.601	0.135
	Range	6 – 16		8.10 ± 2.94			
Gender	Male	26	74.3	17	48.6	X^2=3.759	0.051
	Female	9	25.7	18	51.4		
Order of Birth	1st	9	25.7	9	25.7	X2=2.800	0.592
	2nd	10	28.6	10	28.6		
	3rd	12	34.3	8	22.9		
	4th	2	5.7	6	17.1		
	5th	2	5.7	2	5.7		
Consanguinity	Yes	13	37.1	10	28.6	X^2=0.583	0.445
	No	22	62.9	25	71.4		
Residency	Urban	34	97.1	30	85.7	X^2=1.547	0.320
	Rural	1	2.9	5	14.3		

Mean systolic and diastolic blood pressure were both significantly higher in asthmatics (112.1 ± 7.48 mmHg and 70.5 ± 8.05 mmHg, respectively) compared to controls (104.0 ± 6.51 mmHg and 62.7 ± 8.81 mmHg), with *p-values* <0.001 for both measures but both were within the normal range according to their ages. Additionally, a higher percentage of asthmatic patients had blood pressure in the higher percentile range (50th to 90th), further indicating significant differences in blood pressure centiles between the groups (*p-value* <0.001).

Table 2: Blood pressure comparison between asthmatic patients and control.

		Group A Asthmatic (Number=35)		Group B Control (Number=35)		Test value	*P-value*
		N	%	N	%		
Systolic blood pressure (mmHg)	Mean ±SD	112.1 ± 7.48		104.0 ± 6.51		T=4.787	0.00**
	Range	100 – 120		100 – 120			
Diastolic blood pressure (mmHg)	Mean ±SD	70.5 ± 8.05		62.7 ± 8.81		T=3.852	0.00**
	Range	60 – 80		50 – 80			
blood pressure to age (percentile)	between 5^{th} and 50^{th}	3	8.6	25	71.4	X^2=9.658	0.00**
	between 50^{th} and 90^{th}	32	91.4	10	28.6		

The majority of the patients had comorbid atopic conditions as 57.1% of asthma group had allergic rhinitis and 45.7% had atopic dermatitis, with various exposures to irritants such as dust, smoke and perfumes. 68.6% having a family history of bronchial asthma.

Total serum IgE concentrations had been significantly greater in asthma group. Interestingly, total serum IgE levels insignificantly differ among the different level of control groups (*p-value* = 0.729), despite the large variability observed especially in the partially controlled group (*p-value* = 0.729).

ROC curve analysis of total serum IgE in asthmatic individuals and controls, a cutoff point set at 400 demonstrated a strong diagnostic performance, reflected by an area under the curve (AUC) of 0.897, demonstrating a high level of accuracy in distinguishing between the two groups. The Specificity was 84.6%, sensitivity was 100%. The positive predictive value was 69.2%, while the negative predictive value was 100%.

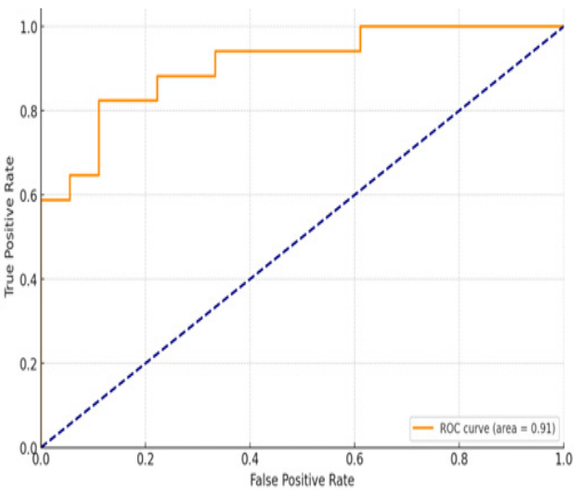

Fig. 1: ROC Curves for IgE in asthmatic and healthy children.

Table 3: Serum IgE level in asthmatic and healthy children.

	Cutoff point	AUC	Sensitivity	Specificity	+PV	-PV
Total serum IgE (IU/ml)	400	0.897	100	84.6	69.2	100

Serum anti-elastin antibodies levels were significantly higher in the asthma group compared to the control group (69.11 ± 63.18 pg/ml vs 6.61 ± 3.68 pg/ml, respectively) with *P-value* < 0.001. The mean levels were significantly higher in uncontrolled patients (91.16 ± 102.58 pg/ml) compared to those in partially controlled (68.85 ± 53.88 pg/ml) and controlled (51.05 ± 44.82 pg/ml) groups, with a highly significant *p-value* (<0.001). Interestingly, serum anti-elastin antibodies showed increased values in patients with blood pressure percentile from 50^{th} to 90^{th} percentile over patients with blood pressure percentile from 5^{th} to 50^{th} percentile.

Table 4: Comparison between asthma and control as regards serum anti-elastin antibodies.

		Group A Asthmatic (Number=35)	Group B Control (Number=35)	Test value	*P-value*
Serum anti-elastin antibodies (pg/ml)	Mean ±SD	69.11 ± 63.18	6.61 ± 3.68	5.842	0.000**
	Range	9.63 – 240	1.95 – 20.55		

Table 5: Comparison between different asthma control levels as regards serum anti-elastin antibodies and total serum IgE.

		Uncontrolled (Number=6)	Partially controlled (Number=22)	Controlled (Number=7)	Test value	*P-value*
Serum anti-elastin antibodies (pg/ml)	Mean±SD	91.16 ± 102.58	68.85 ± 53.88	51.05±44.82	11.069	<0.001**
	Range	17.99 – 240	14.53 - 240.0	9.63 - 156.7		
Total serum IgE (IU/ml)	Mean±SD	342.5 ± 282.75	516.27 ± 662.00	363.14±334.17	0.319	0.729
	Range	100 – 840	10 – 3000	50 - 1117		

The ROC curve analysis of anti-elastin antibodies, demonstrated a high diagnostic performance in differentiating between asthmatic and control groups. The AUC of 0.954 indicated excellent accuracy of the test. The selected cutoff point of 14.8 yielded a specificity of 97.0% a and sensitivity of 96.6%. Positive predictive value of 96.9% and negative predictive value of 97.1%, But moderate ability to differentiate among different levels of asthma control, with an AUC of 0.489. The optimal cutoff point of 47.31 provided specificity of 71.4% and sensitivity of 46.4%. Negative predictive value of 25.0%, and positive predictive value of 86.7%.

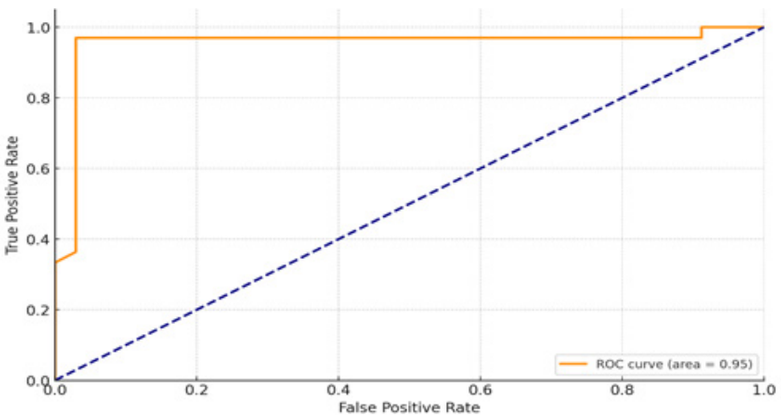

Fig. 2: ROC curve for anti-elastin antibodies to differentiate among healthy and asthmatic children.

Table 6: Serum anti-elastin antibodies level to differentiate between healthy and asthmatic children.

	Cutoff point	AUC	Sensitivity	Specificity	+PV	-PV
Anti-elastin antibodies	14.8	0.954	96.6	97.0	96.9	97.1

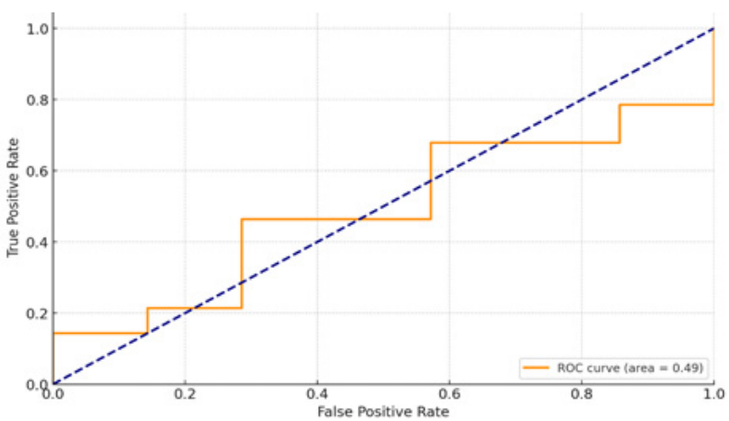

Fig. 3: ROC curves for anti-elastin antibodies to differentiate between different levels of asthma control.

Table 7: Serum anti-elastin antibodies level to differentiate between different levels of asthma control.

	Cutoff point	AUC	Sensitivity	Specificity	+PV	-PV
Anti-elastin antibodies (pg/ml)	47.31	0.489	46.4%	71.4%	86.7%	25.0%

Table 8: Comparison between asthmatic patients and control as regards right and left carotid intima-media thickness.

		Group A Asthmatic (Number=35)	Group B Control (Number=35)	Test value	*P-value*
Average right side (cm)	Mean ±SD	0.050 ± 0.008	0.048 ± 0.007	0.957	0.342
	Range	0.04 – 0.07	0.03 – 0.07		
Percent of change in right intima-media thickness	Mean ±SD	4.0 ± 0.13 %			
	Range	3.85 – 4.15 %			
Average left Side (cm)	Mean ±SD	0.051 ± 0.007	0.048 ± 0.009	0.163	0.163
	Range	0.04 – 0.07	0.03 – 0.07		
Percent of change in left intima-media thickness	Mean ±SD	5.8 ± 0.22 %			
	Range	5.4 – 6.0 %			

The outcomes demonstrated a weak positive correlation among anti-elastin antibodies and both right and left carotid intima-media thickness (right: r = 0.220, $p = 0.067$), (left: r = 0.162, $p = 0.181$) but this was statistically insignificant. Despite this, the strong positive correlation between serum IgE concentrations and anti-elastin antibodies (r = 0.774, $p < 0.001$) was highly significant, suggesting a link between allergic responses and systemic vascular changes in asthmatic children.

Table 9: Comparison between blood pressure to age (percentile) groups as regards serum anti-elastin antibodies and CIMT.

		Blood Pressure to age (Percentile)				Test value	*P-value*
		between 5 and 50 (Number=28)		between 50 and 90 (Number=42)			
		N	%	N	%		
Serum anti-elastin antibodies (pg/ml)	Mean ±SD	12.8 ± 21.19		54.6 ± 63.08		3.370	0.001**
	Range	2.25 – 109.5		1.95 – 240			
Right carotid intima-media thickness (cm)	Mean ±SD	0.048 ± 0.008		0.049 ± 0.008		0.135	0.893
	Range	0.03 – 0.07		0.03 – 0.07			
Left carotid intima-media thickness (cm)	Mean ±SD			0.050 ± 0.008		0.477	0.635
	Range	0.03 – 0.07		0.03 – 0.07			

Regarding Carotid intima-media thickness, our research outcomes discovered that significant percent of increase (4%) was found in right carotid intima-media thickness and (5.8%) in left side in asthmatic over control groups. Similarly, both left and right CIMT measurements did not show significant differences neither across the asthma control levels, with *p-values* of 0.775 for the right carotid and 0.139 for the left, nor across different blood pressure percentile groups.

DISCUSSION

Recent evidence indicates a correlation between asthma and atherosclerosis as well as cardiovascular disease. Nonetheless, data concerning kids and adolescents are limited and conflicting[8].

Since asthma in children represents major conflict and may be associated with cardiovascular complications,

evaluating the effect of asthma on carotid intima-media thickness in a significant pediatric population was emphasized as a primary focus[9].

The exact pathways linking inflammation in asthma and atopic diseases to arterial damage and elevated cardiovascular disease (CVD) risk remained undetermined. In childhood asthma, there is often an imbalance in T-helper cell polarization, which manifests primarily in a Type 1 (T1) or Type 2 (T2) inflammatory response[5].

In our results, we classified our patients to 62.9% (N=22) as partially controlled, indicating that while their asthma was managed to some extent, there might still be symptoms affecting their daily life, 20.0% (N=7) of patients had their asthma fully controlled and 17.1% (N=6) of patients remained uncontrolled.

Regarding blood pressure, our results showed that systolic and diastolic blood pressure had been both significantly higher in asthmatics compared to controls, but both were within the normal range according to their ages. Additionally, a higher percentage of asthmatic patients had blood pressure in the higher percentile range (50^{th} to 90^{th}), further indicating significant differences in blood pressure profiles between the groups (*p-value* <0.001).

In agreement with our outcomes, ***Winder et al.***, (2022) conducted the community-based early vascular ageing-Tyrol cohort study and found that allergic asthmatics demonstrated higher blood pressure concentrations than healthy controls[9]. ***Dogra et al.***, (2010) and ***Aung et al.***, (2010) similarly documented these results, indicating a heightened frequency of hypertension in adults with asthma or inhalative allergies[10,11] Nonetheless, this correlation was not evident in all investigations, especially those involving pediatric groups[5, 12].

The mechanism of elevated blood pressure was explained mainly by smooth-muscle remodeling triggered by inflammatory mediators. Hyperplasia and abnormal contraction of smooth muscle cells leading to airway obstruction in asthma have been described as characteristics of vascular remodeling and endothelial abnormalities[9].

Serum anti-elastin antibodies were significantly higher in asthma group compared to control group (69.11 ± 63.18 pg/ml vs 6.61 ± 3.68 pg/ml, respectively) with *P-value* <0.001, and when comparing between blood pressure to age (percentile) groups and serum anti-elastin antibodies, it was found that serum anti-elastin antibodies demonstrated a significant variance (*p-value* = 0.001) among both groups, with a higher mean value in the group between 50^{th} and 90^{th} percentiles (54.6 ± 63.08 pg/ml) compared to the group between 5^{th} and 50^{th} percentiles (12.8 ± 21.19 pg/ml), indicating a strong association with blood pressure levels. This was attributed to the accumulation of elastin beneath the basement membrane around the bronchi, leading to airway remodeling, which was more prominent in asthma patients than in controls. The reasons for the elevated anti-elastin antibody levels in asthma patients remain unclear, and no reports were found linking these factors[8].

To explore the targets of anti-elastin antibodies, ***Tokita et al.,*** (2021) conducted staining of lung tissue with anti-elastin antibodies. Despite their efforts, detecting anti-elastin antibodies in lung tissue proved challenging[8]. They proposed two conflicting hypotheses regarding the role of anti-elastin antibodies.

One hypothesis suggested that these antibodies were produced to counteract the increase in elastin and prevent airway remodeling. The other hypothesis posited that anti-elastin antibodies contributed to airway remodeling through a severe inflammatory response triggered by an antigen-antibody reaction to elastin beneath the basement membrane of the bronchi. The study further investigated the role of anti-elastin antibodies using a mouse model of asthma, finding a significant increase in anti-elastin antibodies in the bronchoalveolar lavage fluid of the asthma model[13].

Regarding Carotid intima-media thickness, our study results revealed that there was (4%) increase in right carotid intima-media thickness and (5.8%) increase in left side in asthmatic over control groups. Similarly, both left and right CIMT measurements did not show significant differences across the asthma control levels, and did not show significant difference between blood pressure to age (percentile) groups.

Supporting these findings several mechanisms might contribute to the arterial injury and elevated cardiovascular disease risk associated with the inflammation seen in early-onset asthma and atopy[5].

This could be explained as childhood asthma and atopic conditions typically exhibited a predominant T2 inflammatory response. This T2 high asthma was marked by increased concentrations of IL-4, a cytokine that promoted atherosclerosis. IL-4 activated the vascular endothelium, facilitating the activation and recruitment of mononuclear cells, boosting the expression of cellular adhesion molecules, and stimulating the production of 15-lipoxygenase, which could oxidize low-density lipoproteins. Furthermore, mast cells, crucial in atopic and asthma conditions, produce cysteinyl leukotrienes (D4, C4, and E4) and trigger both chronic and acute inflammation[5].

Additionally, arterial injury and subsequent atherosclerosis developed rapidly in the carotid bulb than in the common carotid artery, attributable to the bifurcation's anatomy and flow disturbances that create areas of low and oscillatory shear stress[14].

Tattersall et al., (2018) conducted a Childhood Origins of Asthma (COAST) Cohort study that enrolled 89 children (twenty-eight asthmatics with atopic illness, thirty-four asthmatics without other atopic illness, 15 non-asthmatics with atopic illness and 12 controls) to investigate the associations between the presence of atopic and asthma disease in kids with greater arterial injury established as a thicker CIMT, compared to control group and revealed that asthmatic kids with or without other atopic illness had thicker right carotid bifurcation (RCB) and right common carotid artery (RCCA) walls than non-asthmatic/non-atopic kids, demonstrating indication of arterial injury, that is antecedent to the progress of cardiovascular disease[5].

Consequently, the early development of significantly thicker carotid bifurcation IMT in these asthmatic and atopic kids detected an early indicator of subclinical arterial alterations. This investigation identified clinically significant variations, as CCA IMT variations of 30±50 micrometers correlated with an estimated 4±6% rise in myocardial infarction and stroke incidence over a ten-year period in adults[5].

To the best of our knowledge, there is a paucity of investigations in literature evaluating the serum anti-elastin antibodies and carotid intima-media thickness in asthmatic kids compared to healthy children.

CONCLUSION

Serum Anti-elastin antibodies as vascular inflammatory marker was higher in asthmatic children compared to control. This may require periodic follow up of any vascular changes in those patients.

Study limitations

1. Small Sample Size: The study included only 70 children (35 asthmatic and 35 controls), which may limit the generalizability of the findings.

2. Single-Center Study: The research was conducted at a single hospital (Children's Hospital, Ain Shams University), which may not be representative of the broader population.

3. Cross-Sectional Design: Since this is a case-control study, it can only establish associations and not causation between asthma, vascular changes, and serum anti-elastin antibodies.

4. Lack of Longitudinal Data: There is no follow-up to assess whether the observed vascular changes progress over time in asthmatic children.

5. Measurement Limitations: Carotid intima-media thickness (CIMT) was measured by ultrasound, which may have some inter-operator variability despite being conducted by the same examiner.

CONFLICT OF INTEREST

1. No competing interests of financial or personal nature.

2. The manuscript is not under consideration elsewhere.

3. Funding not received .

REFERENCES

1. **Calvén J, Ax E, Rådinger M.** The airway epithelium A central player in asthma pathogenesis. Int. J. Mol. Sci. 2020; 21(23):8907.

2. **Karlöf E, Seime T, Dias N, Lengquist M, Witasp A, *et al.*** Correlation of computed tomography with carotid plaque transcriptomes associates calcification with lesion-stabilization. ASND. 2019; 288:175-85.

3. **Joglekar MM, Bekker NJ, Koloko Ngassie ML, Vonk JM, Borghuis T, *et al.*** The lung extracellular matrix protein landscape in severe early-onset and moderate chronic obstructive pulmonary disease. American Journal of Physiology-Lung Cellular and Molecular Physiology. 2024; 327(3):L304-18.

4. **Drole Torkar A, Plesnik E, Groselj U, Battelino T, Kotnik P.** Carotid intima-media thickness in healthy children and adolescents: normative data and systematic literature review. Frontiers in Cardiovascular Medicine. 2020; 7:597768.

5. **Tattersall MC, Evans MD, Korcarz CE, Mitchell C, Anderson E, *et al.*** Asthma is associated with carotid arterial injury in children: The Childhood Origins of Asthma (COAST) Cohort. PLoS One. 2018; 13(9):e0204708.

6. **2023 GINA Main Report - Global Initiative for Asthma - GINA.** Global Initiative for Asthma - GINA. https://ginasthma. org/2023-gina-main-report. 2023.

7. **Cakmak A, Zeyrek D, Cece H, Erel O.** The relationship between carotid intima media thickness and oxidative stress in asthmatic children. APJAI. 2010; 28(4):256.

8. **Tokita S, Sugiyama K, Wakayama T, Arifuku H, Otsuji N, *et al.*** Elevation of anti-elastin antibody in patients with asthma. APJAI. 2021;10. 12932/AP-010221-1052.

9. **Winder B, Kiechl SJ, Gruber NM, Bernar B, Gande N, *et al.*** The association of allergic asthma and carotid intima-media thickness in adolescence: data of the prospective early vascular ageing (EVA)-Tyrol cohort study. BMC Cardiovascular Disorders. 2022; 22(1):11.

10. **Dogra S, Ardern CI, Baker J.** The relationship between age of asthma onset and cardiovascular disease in Canadians. J Asthma. 2007;44(10):849–54.

11. **Aung T, Bisognano JD, Morgan MA.** Allergic respiratory disease as a potential co-morbidity for hypertension. Cardiol J. 2010;17(5):443–7.

12. **Roelofs R, Gurgel RQ, Wendte J, Polderman J, Barreto-Filho JA, *et al***. Relationship between asthma and high blood pressure among adolescents in Aracaju. Brazil J Asthma. 2010;47(6):639–43

13. **Wakayama T, Sugiyama K, Tokita S, Arifuku H, Otsuji N, *et al.*** The role of anti-elastin antibodies in a mouse model of asthma. Asian Pacific Journal of Allergy and Immunology. 2021.

14. **Tzou WS, Douglas PS, Srinivasan SR, Bond MG, Tang R, *et al.*** Distribution and predictors of carotid intima-media thickness in young adults. Prev Cardiol. 2007; 10(4):181-9.

الأجسام المضادة للإيلاستين في الدم وسمك الطبقة الداخلية للشريان السباتي كمؤشرات على المؤثر الوعائي عند الأطفال المصابين بالربو الشعبى

ايمان محمود فودة[١]، ماريز زكريا عدلى[٢]، بسمة شريف فهمي[٣]، اسعد جمال اسعد[٤] و هبة مصطفى حمزة[١]

[١]وحده امراض الرئه، [٢]مستشفى الاطفال، [٣]قسم الباثولوجيا الأكلينيكية، [٤]قسم الاشعة التشخيصية و التداخلية و التصوير الجزيئي، كلية الطب، جامعة عين شمس

المقدمة: ارتباط الربو بزيادة خطر الإصابة بأمراض القلب والأوعية الدموية، على الأرجح بسبب وجود آلية التهابية مشتركة.

الهدف: تهدف هذه الدراسة إلى تقييم الأجسام المضادة للإيلاستين وسمك الشرايين السباتية كتغيرات وعائية فى الأطفال المصابين بالربو وعلاقتها بمستوى شدة الربو والسيطرة عليه.

المرضى والطرق: اجريت هذه الدراسة المستقبلية للحالات في وحدة أمراض الصدر بمستشفى الأطفال، جامعة عين شمس، وأُجريت على إجمالي ٣٥ طفلًا مصابين بالربو مقارنة بـ ٣٥ طفلًا صحيًا من نفس العمر والجنس. تم إخضاعهم لأخذ تاريخ مرضي مفصل، والفحص الشامل والفحوصات التالية:

- صورة الدم الكاملة
- بروتين سي التفاعلي
- مستوى الغلوبولين المناعي ه
- الأجسام المضادة للإيلاستين
- قياس سمك الشريان السباتي

النتائج: كان مستوى الأجسام المضادة للإيلاستين أعلى بشكل ملحوظ لدى الأطفال المصابين بالربو مقارنةً بمجموعة الضبط. كما كان المستوى أعلى إحصائيًا لدى الأطفال المصابين بالربو غير المسيطر عليه مقارنةً بالحالات المسيطر عليها والحالات المسيطر عليها جزئيًا.

وُجدت علاقة إيجابية ذات دلالة إحصائية بين مستوى الغلوبولين المناعي ه ومستوى الأجسام المضادة للإيلاستين.

أما سمك الشريان السباتي لدى الأطفال المصابين بالربو فقد وجد زيادة نسبتها٤٪ في الشريان السباتي الأيمن و٥,٤٪ في الأيسر. لم يكن هناك فرق ذو دلالة إحصائية في متوسط سمك الشريان السباتي بين الأطفال المصابين بالربو باختلاف مستوى السيطرة على المرض

الاستنتاج: كانت مستويات الأجسام المضادة للإيلاستين، كمؤشر للالتهاب الوعائي، أعلى لدى الأطفال المصابين بالربو مقارنةً بالمجموعة الضابطة، مما قد يستدعي متابعة دورية لأي تغييرات وعائية لدى هؤلاء المرضى.