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ORIGINAL ARTICLE

Right ventricular function in children with asthma: tissue doppler and two-dimensional speckle-tracking echocardiography

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Abstract
Background: Asthma-related cardiopulmonary interactions may affect right ventricular (RV) performance even when children are clinically stable. Two-dimensional speckle-tracking echocardiography (2D-STE) may detect subclinical myocardial dysfunction beyond conventional indices. This study aims to investigate the effects of childhood asthma on RV function by using 2D-STE.
Methods: In this prospective case-control study, children aged 6-18 years with physician-diagnosed asthma (n = 70) and age-matched healthy controls (n = 30) underwent standard transthoracic echocardiography, tissue Doppler imaging (TDI), and 2D-STE. Primary outcomes were RV global longitudinal strain (RV GLS) and strain rate. Conventional RV systolic indices and RV diastolic indices were also assessed. Clinical characteristics and spirometry were recorded.
Results: RV systolic mechanics were largely preserved in the asthma group. RV GLS, RV strain rate, and tricuspid annular plane systolic excursion (TAPSE) did not differ significantly between asthmatic children and controls. In contrast, tricuspid annular E′ velocity was significantly lower in the asthma group (p = 0.007), and tricuspid E/E′ ratio was significantly higher (p < 0.001). Interventricular septal diastolic thickness was also significantly increased among children with asthma (p = 0.027). Within the asthma cohort, TAPSE showed positive correlations with age (r = 0.478, p < 0.001), BMI (r = 0.398, p = 0.001), asthma duration (r = 0.431, p < 0.001), and FEV1 (r = 0.301, p = 0.011), whereas RV strain parameters were not significantly associated with demographic or respiratory variables.

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Conclusions: In predominantly mild-to-moderate, clinically stable pediatric asthma, RV systolic deformation by 2D-STE appears preserved, while TDI-derived indices indicate early RV diastolic involvement. Focused assessment of RV diastolic function may help to identify subclinical cardiopulmonary effects in selected children with asthma.

Introduction

Asthma is the most common chronic respiratory disease in childhood, characterized by attacks of wheezing, shortness of breath, and coughing.[1,2] It is associated with high morbidity and has a significant impact on quality of life.[3-5] Bronchial hypersensitivity and airway inflammation, which result in airflow limitations and hypoxia attacks, are responsible for episodes of respiratory symptoms. Recurrent attacks of hypoxemia and airway inflammation cause pulmonary artery vasoconstriction and increased pulmonary vascular resistance and may lead to right ventricular (RV) dysfunction and pulmonary hypertension (PH) over time.[6,7] The negative impact of chronic respiratory diseases such as asthma on cardiovascular function has been reported in the literature.[8-11]

Conventional echocardiography is a noninvasive, widely available, and fast method for assessing cardiovascular function. Because assessment of RV function is relatively complex by this method, techniques such as Tissue Doppler imaging (TDI) and two-dimensional speckle-tracking echocardiography (2D-STE) have been used to detect subclinical and early cardiovascular dysfunction in patients with asthma.[10,12-14] These techniques enable quantification of myocardial deformation parameters such as strain and strain rate, which provide insight into systolic and diastolic dysfunction, ischemia, and myocardial mechanics.[15] Compared with TDI, 2D-STE mitigates several limitations such as angle dependence and frame-rate constraints.[15] Although 2D-STE has been reported as useful for the early detection of cardiovascular dysfunction in chronic respiratory disease, pediatric data in asthma remain limited.[11,12,16]

Therefore, we aimed to evaluate RV function in children with asthma using conventional echocardiography and 2D-STE, and to compare these findings with those of healthy controls.

Materials and Methods

Study design and participants

This single-center prospective case–control study was conducted at the Pediatric Allergy Outpatient Clinic, Istanbul University Faculty of Medicine. Seventy children with asthma aged 6–18 years who had not experienced an asthma attack within the previous month and had no chronic disease other than asthma except allergic comorbidities were included. The control group consisted of 30 healthy children aged 6–18 years without a history of atopy or any chronic disease.

Asthma diagnosis and clinical assessment

Asthma was diagnosed in accordance with the Global Initiative for Asthma (GINA) guidelines, based on characteristic clinical symptoms and good response to asthma therapy and/or objective evidence of variable expiratory airflow limitation. Asthma control was assessed using the Childhood Asthma Control Test (c-ACT) in children aged ≤11 years and the Asthma Control Test (ACT) in those aged ≥12 years.[17-19] Asthma severity was classified according to the GINA-based clinical assessment, taking into account symptom control, exacerbation history, medication intensity, and treatment step. The clinical phenotype was characterized by allergic comorbidities, atopy, skin prick test positivity, eosinophil percentage, total IgE level, asthma control status, and current controller treatment.

Demographic data, spirometry results (forced expiratory volume in 1 second [FEV1], peak expiratory flow [PEF], and FEV1/forced vital capacity [FVC]), total immunoglobulin E (IgE), and eosinophil levels were recorded for children with asthma. Atopy was defined as evidence of allergic sensitization, whereas concomitant allergic disease was recorded as a composite clinical variable referring to physician-diagnosed allergic comorbidities accompanying asthma, such as allergic rhinitis, atopic dermatitis, and food allergy. Weight, height, and arterial blood pressure were measured in all participants. Arterial blood pressure was measured by auscultation using an age-appropriate cuff after 20 minutes of rest.

Conventional echocardiography

All participants (n = 100) underwent conventional echocardiographic assessment. Echocardiographic examinations were performed according to standard images and techniques as recommended by the American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI) guidelines.[20,21] The echocardiographer was blinded to participant group allocation during image acquisition and analysis. Echocardiography was performed after at least 15 minutes of rest in the left lateral position (2-dimensional, M-mode, Doppler echocardiography) from the parasternal and apical windows using the Philips iE33 device and the X5-1 transthoracic probe. M-mode images were obtained between the mitral valve and papillary muscle on the parasternal long axis. Care was taken to ensure that the ultrasound beam hit the interventricular septum (IVS) and the posterior wall of the left ventricle (LV) perpendicularly. In this way, the diameters of the LV during diastole and systole were visible. Diastolic and end-systolic internal diameters were measured from the extreme points of the endocardium, and left ventricular ejection fraction (EF) was calculated using the Teicholz method.[22]

RV diastolic function was assessed using pulsed-wave Doppler and TDI. Transtricuspid diastolic inflow velocities were obtained by positioning the pulsed-wave Doppler sample volume at the tips of the tricuspid valve leaflets in the apical four-chamber view. Peak early (E) and late (A) diastolic velocities were measured, and the E/A ratio was calculated.

TDI was used to assess tricuspid annular velocities. Early (E′) and late (A′) diastolic myocardial velocities were measured at the lateral tricuspid annulus in the apical four-chamber view, and the E/E′ ratio was calculated as an estimate of RV filling pressure.

Tricuspid annular plane systolic excursion

Tricuspid annular plane systolic excursion was obtained with M-mode tracing from where the tricuspid annulus meets the lateral free wall in the apical four-chamber view. Most RV movement occurs through subendocardial myocardial fibers located longitudinally in the thin RV wall. For this reason, it has been shown that the tricuspid annulus movement, which occurs between the annular plane and the apex in the long axis, provides information about global RV functions and is correlated with RV EF.[23] This parameter, which is easy and fast to apply, is preferred over other two-dimensional parameters in terms of RV systolic functions.

Two-dimensional speckle tracking echocardiography (2D-STE)

Global longitudinal strain (GLS) and strain rate measurements were performed on all participants using the 2D-STE method. Philips iE33 and QLAB-CMQ software were used in the analyses. Measurements were obtained from apical 4-chamber images containing at least 4 cardiac cycles, with a frame rate of 70–100 frames/s. A separate dedicated RV-focused apical four-chamber acquisition was not systematically performed. First, the boundaries of the RV endocardium were drawn, and then, based on the model recommended by the ASE/EACVI guidelines, peak systolic strain and peak systolic strain rate measurements were automatically made in each segment through the analysis program from these records. RV GLS included both septal and RV free-wall segments; RV free-wall strain was not analyzed separately (Figure 1).

Statistical analysis

SPSS software (Statistical Package for Social Sciences), version 24.0 was used for statistical data analysis. The normality of continuous variables was assessed using the Shapiro–Wilk test and visual inspection of histograms. Descriptive data were presented as mean ± standard deviation or median, minimum, maximum, as appropriate. For comparisons between two independent groups, Student's *t* test was used for normally distributed variables, and the Mann–Whitney *U* test was used for non-normally distributed variables. For comparisons involving more than two groups, one-way analysis of variance (ANOVA) was used when normality assumptions were met, whereas the Kruskal–Wallis test was used when these assumptions were not met. Chi-square test was used for categorical variables. No correction for multiple comparisons was applied, and subgroup analyses were interpreted as exploratory.Correlation analyses were performed using Pearson or Spearman tests, as appropriate. The results were evaluated with a 95% confidence interval, with significance accepted at $p < 0.05$.

Ethical approval

Ethical approval for the study was granted by the Istanbul University Ethics Committee (No: 1538). The study was conducted in accordance with the principles of the World Medical Association Declaration of Helsinki. Written informed consent was obtained from the parents or legal guardians of all participants, and assent was obtained from children when appropriate.

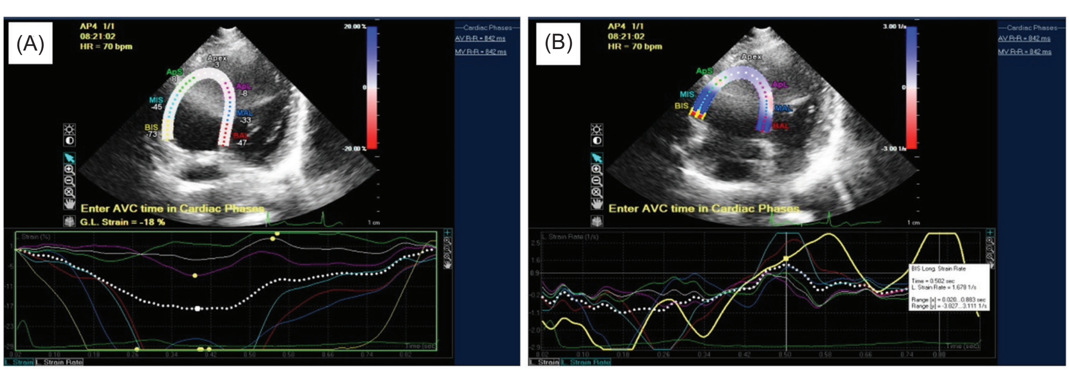

Figure 1 Right ventricle peak systolic strain (A) and strain rate (B) measurements in 2D-STE. RV strain reflects the degree of longitudinal myocardial deformation during systole, whereas strain rate reflects the speed of this deformation. These parameters allow quantitative assessment of RV systolic mechanics and may help detect subtle myocardial dysfunction before abnormalities become evident on conventional echocardiographic indices.

Results

A total of 70 children with asthma and 30 healthy controls were included in the study. Among the children with asthma, 52.9% (n = 37) were female, and the mean age at the last examination was 11.4 ± 2.9 years (range, 6.5–17 years). In the control group, 43.3% (n = 13) were female, with a mean age of 10.9 ± 3.2 years (range, 6–17.8 years). The mean body mass index (BMI) was 20.3 ± 4.6 kg/m^2 (range, 11.9–33.2) in children with asthma and 18.7 ± 4.4 kg/m^2 (range, 13.8–29.2) in healthy controls. There were no significant differences between the two groups with respect to sex, age, or BMI ($p > 0.05$). Arterial blood pressure values were within the normal range in both groups.

In children with asthma, the median age at diagnosis was 3.0 years (IQR, 2.0–5.0). The median follow-up duration was 7.0 years (IQR, 4.8–9.6). The median time since the last asthma attack was 6.0 months (IQR, 3.0–12.0). Spirometric evaluation showed a mean FEV1 of 92.1 ± 12.3% predicted (range, 64–126), a mean FEV1/FVC ratio of 111.1 ± 7.3% predicted (range, 89–119), and a mean PEF of 86.5 ± 14.4% predicted (range, 56–137). One patient was classified as having severe asthma, while the remaining patients had mild to moderate asthma. Detailed demographic, clinical, and laboratory characteristics of the children with asthma are summarized in Table 1.

Table 1 Demographic, clinical, and laboratory data of asthmatic children (n = 70).

Demographic and Clinical Characteristics	n (%)
Birth (preterm)	10 (14.3)
Maternal smoking during pregnancy	10 (14.3)
Consanguinity	8 (11.4)
Atopy in the family	54 (77.1)
Family history of heart disease (atherosclerosis, valve disease, arrhythmia, etc.)	41 (58.6)
Passive smoking	28 (40)
Concomitant allergic disease	53 (75.7)
Treatment	
ICS	15 (21.4)
ICS + LTRA	26 (37.4)
ICS + LABA	20 (28.4)
ICS + LABA + LTRA	9 (12.9)
Asthma control test	
Well controlled	15 (21.4)
Partly controlled	47 (67.1)
Uncontrolled	8 (11.4)
Skin prick test positivity	
Any aeroallergen sensitization	49 (70)
House dust mite sensitization[a]	16 (22.9)
Total IgE kU/L (≥100)	53 (75.7)
Eosinophil (%) (≥4)	32 (45.7)
Atopy	60 (85.7)

ICS, inhaled corticosteroid; LABA, long-acting beta agonist; LTRA, leukotriene receptor antagonist.
[a]House dust mite sensitization is presented as a subgroup of aeroallergen sensitization; percentages were calculated using the total asthma cohort as the denominator.

Conventional echocardiographic and 2D-STE parameters were compared between children with asthma and healthy controls (Table 2). Left ventricular dimensions, posterior wall thickness, EF, RV and right atrial diameters, transtricuspid inflow velocities (E and A), tricuspid annular A′ velocity, TAPSE, and RV GLS and strain rate were similar between groups (all p > 0.05). Interventricular septal diastolic thickness was significantly greater in children with asthma than in controls ($p = 0.027$). In addition, tricuspid annular early diastolic velocity (E′) was significantly lower in the asthma group ($p = 0.007$), whereas the tricuspid E/E′ ratio was significantly higher ($p < 0.001$). No significant difference was observed in transtricuspid E/A and E'/A' ratio between groups ($p > 0.05$).

Within the asthma group, RV strain, strain rate, and TAPSE were analyzed according to clinical and laboratory characteristics (Table 3). Overall, no significant differences were observed in strain, strain rate, or TAPSE across subgroups defined by sex, birth status (preterm vs term), maternal smoking during pregnancy, concomitant allergic disease, family history of atopy, passive smoking exposure, skin prick test positivity, total IgE category (<100 vs ≥100 IU/mL), eosinophil percentage (<4% vs ≥4%), atopy status, asthma treatment modality, or asthma control level ($p > 0.05$).

In children with asthma, strain and strain rate were not significantly correlated with age, BMI, asthma duration, time since the last asthma attack, FEV1, or PEF ($P > 0.05$; Table 4). In contrast, TAPSE showed weak-to-moderate positive correlations with age ($r = 0.478$, $p < 0.001$), BMI ($r = 0.398$, $p = 0.001$), asthma duration ($r = 0.431$, $p < 0.001$), and FEV_1 ($r = 0.301$, $p = 0.011$), while no significant correlation was observed with time since the last asthma attack ($r = 0.076$, $p = 0.530$) or PEF ($r = 0.200$, $p = 0.097$).

Discussion

In this prospective case–control study, RV systolic function markers were evaluated in children with asthma aged 6–18 years using conventional echocardiography and 2D-STE. We found no significant differences between asthmatic and healthy children in RV GLS, strain rate, and TAPSE. However, IVS diastolic thickness and the tricuspid E/E′ ratio were higher, while tricuspid annular E′ was lower in the asthma group, suggesting subtle diastolic involvement despite preserved systolic deformation.

Our finding of increased IVS diastolic thickness may align with emerging evidence of subtle structural remodeling in asthma. In the BADA (Blood Pressure Levels, Clinical Features, and Markers of Subclinical Cardiovascular Damage of Asthma Patients) study ECO substudy, Di Raimondo et al. reported greater IVS thickness in children with asthma than in controls, supporting the concept that long-standing asthma may be associated with subclinical cardiac remodeling.[24] Although pediatric and adult populations differ substantially and direct extrapolation should be cautious, these data provide a biologically plausible context in which repeated increases in pulmonary vascular tone and RV loading could contribute to septal remodeling over time.

Table 2 Echocardiography parameters of asthmatic and healthy children

	Patient group (mean ± SD)	Control group (mean ± SD)	*p* value
LV diastolic diameter (cm)	4.16 ± 0.53	4.17 ± 0.51	0.936
LV systolic diameter (cm)	2.54 ± 0.39	2.58 ± 0.38	0.628
IVS diastolic thickness (cm)	0.78 ± 0.14	0.71 ± 0.16	**0.027**
LV diastolic posterior wall thickness (cm)	0.73 ± 0.12	0.71 ± 0.17	0.567
EF (%)	69.39 ± 5.00	68.72 ± 5.29	0.556
RV diameter (cm)	2.25 ± 0.37	2.33 ± 0.30	0.288
RA diameter (cm)	2.88 ± 0.49	3.04 ± 0.32	0.066
Tricuspid E (cm/s)	71.83 ± 10.24	74.49 ± 10.55	0.232
Tricuspid A (cm/s)	44.57 ± 9.98	46.79 ± 10.78	0.322
Tricuspid E' (cm/s)	13.66 ± 3.47	15.57 ± 2.87	**0.007**
Tricuspid A' (cm/s)	5.97 ± 1.48	6.24 ± 1.45	0.391
E/A	1.66 ± 0.34	1.64 ± 0.34	0.798
E'/A'	2.62 ± 0.78	2.40 ± 0.76	0.176
E/E'	7.41 ± 1.63	6.14 ± 1.23	**<0.001**
TAPSE (cm)	2.38 ± 0.35	2.25 ± 0.27	0.054
RV GLS (%)	−26.36 ± 2.95	−25.63 ± 3.45	0.321
RV GLS rate (s−1)	−1.57 ± 0.80	−1.79 ± 0.39	0.070

Bold text indicates statistically significant results.
EF, ejection fraction; GLS, global longitudinal strain; IVS, interventricular septum; LV, left ventricle; RA, right atrium; RV, right ventricle; SD, standard deviation; TAPSE, tricuspid annular plane systolic excursion.

These findings should be interpreted in the clinical context of our cohort, which predominantly consisted of mild-to-moderate asthma with generally preserved spirometry and a relatively long interval since the last attack. Although childhood asthma is clinically stable, recurrent hypoxemia, airway inflammation, and episodic increases in pulmonary vascular tone can intermittently create afterload stress on the right ventricle. Such physiology may affect RV relaxation and filling properties earlier than it alters global systolic deformation, producing a "diastolic-first" signal detectable by tissue Doppler indices even when conventional chamber dimensions and systolic indices remain within normal limits.

Our TDI findings are consistent with earlier pediatric studies demonstrating subclinical RV diastolic changes in asthmatic children. In a classic tissue Doppler study, Shedeed reported significantly altered tricuspid annular E′ and related diastolic parameters in children with asthma studied between attacks, supporting the presence of early RV diastolic dysfunction.[14] Similarly, Akyüz Özkan et al. found lower tricuspid E′ and lower TAPSE in asthmatic children compared with controls, suggesting that both diastolic impairment and subtle reductions in longitudinal systolic motion may be present in some cohorts.[25] More recent data using tissue doppler methods have shown that tricuspid annular E′ can be reduced, and RV relaxation indices may worsen with increasing asthma severity, reinforcing the concept that diastolic dysfunction may be an early marker in pediatric asthma.[26] Consistent with this pattern, De-Paula et al. also reported significantly lower tricuspid annular E′ and lower E′/A′ ratio in young patients with asthma compared with controls, supporting impaired RV relaxation even when conventional measures appear normal.[27]

Pediatric strain imaging studies in asthma have reported heterogeneous results, likely driven by differences in severity distribution, asthma control, and methodology. Abdelmohsen et al. preserved global RV longitudinal strain in children with mild-to-moderate asthma despite evidence of RV diastolic dysfunction.[17] In Abbas et al. found lower peak longitudinal systolic strain values in asthmatic children, suggesting that deformation abnormalities may be detectable in cohorts with greater clinical burden.[12] Similarly, Elmeazawy et al. reported lower 2D RV longitudinal strain in children with severe asthma compared with mild/moderate asthma groups, supporting a severity-related gradient in myocardial deformation.[28] Together, these findings suggest that RV strain abnormalities may become more evident as asthma burden increases, whereas preserved RV strain in our cohort is consistent with predominantly stable, mild-to-moderate asthma.

Interpretation of RV strain in children requires careful consideration of age- and vendor-dependent reference values. A meta-analysis defining pediatric normative RV strain values reported mean RV GLS around −29% and mean systolic strain rate around $-1.9\ s^{-1}$, highlighting wide but interpretable normal ranges.[29] Contemporary pediatric studies providing age-specific strain norms and z-score approaches further emphasize the need for platform-aware comparisons when interpreting strain across cohorts and centers.[30] Using these frameworks, the RV strain and strain rate values observed in both our asthmatic and control groups were within expected boundaries, supporting preserved RV systolic function in this population.

Table 3 Strain, strain rate and TAPSE according to clinical and laboratory features in asthmatic children

	Strain (%)		Strain Rate (s^{-1})		TAPSE (cm)	
	Mean ± SD	*P* value	Mean ± SD	*P* value	Mean ± SD	*P* value
Gender						
Male	−26.76 ± 2.80	0.284	−1.53 ± 0.88	0.902	2.42 ± 0.33	0.347
Female	−26.00 ± 3.07		−1.61 ± 0.74		2.34 ± 0.33	
Birth week						
Preterm	−25.20 ± 2.90	0.171	−1.55 ± 0.90	0.926	2.37 ± 0.23	0.706
Term	−26.55 ± 2.94		−1.58 ± 0.79		2.38 ± 0.36	
Maternal smoking during pregnancy						
No	−26.27 ± 2.94	0.464	−1.55 ± 0.80	0.507	2.37 ± 0.34	0.860
Yes	−26.90 ± 3.11		−1.71 ± 0.87		2.40 ± 0.42	
Concomitant allergic disease						
No	−26.94 ± 2.56	0.389	−1.53 ± 0.85	0.789	2.29 ± 0.29	0.215
Yes	−26.17 ± 3.06		−1.59 ± 0.80		2.41 ± 0.36	
Atopy in the family						
No	−27.06 ± 2.57	0.228	−1.70 ± 0.67	0.590	2.38 ± 0.46	0.748
Yes	−26.15 ± 3.04		−1.54 ± 0.84		2.38 ± 0.31	
Family history of heart disease						
No	−27.28 ± 2.67	0.017	−1.75 ± 0.73	0.190	2.44 ± 0.41	0.280
Yes	−25.71 ± 2.99		−1.45 ± 0.84		2.33 ± 0.29	
Passive smoking						
No	−26.36 ± 2.92	0.926	−1.51 ± 0.81	0.476	2.34 ± 0.33	0.328
Yes	−26.36 ± 3.05		−1.67 ± 0.79		2.43 ± 0.36	
Treatment						
ICS	−26.47 ± 2.83	0.410	−1.80 ± 0.73	0.543	2.46 ± 0.36	0.060
ICS+LTRA	−25.81 ± 3.02		−1.49 ± 0.80		2.25 ± 0.28	
ICS+LABA	−27.00 ± 3.01		−1.43 ± 0.89		2.45 ± 0.40	
ICS+LABA+LTRA	−26.33 ± 3.00		−1.75 ± 0.72		2.47 ± 0.32	
Asthma control test						
Well controlled	−27.53 ± 1.68	0.367	−1.80 ± 0.56	0.451	2.41 ± 0.39	0.931
Partly controlled	−26.06 ± 3.17		−1.48 ± 0.86		2.37 ± 0.35	
Uncontrolled	−25.87 ± 3.18		−1.70 ± 0.82		2.36 ± 0.29	
Skin prick test						
Negative	−26.48 ± 2.80	0.952	−1.60 ± 0.85	0.778	2.36 ± 0.26	0.724
Positive	−26.31 ± 3.04		−1.57 ± 0.79		2.39 ± 0.38	
Total IgE						
<100	−25.82 ± 3.23	0.526	−1.53 ± 0.90	0.924	2.43 ± 0.32	0.317
≥100	−26.53 ± 2.87		−1.59 ± 0.78		2.36 ± 0.36	
Eosinophil (%)						
<4	−26.39 ± 3.12	0.908	−1.52 ± 0.87	0.544	2.40 ± 0.35	0.502
≥4	−26.31 ± 2.79		−1.64 ± 0.72		2.35 ± 0.34	
Atopy						
No	−26.50 ± 2.42	0.986	−1.64 ± 0.82	0.854	2.44 ± 0.24	0.208
Yes	−26.33 ± 3.05		−1.56 ± 0.81		2.37 ± 0.36	

ICS, inhaled corticosteroid; LTRA, leukotriene receptor antagonist; LABA, long-acting beta agonist TAPSE, tricuspid annular plane systolic excursion. Bold text indicates statistically significant results.
Strain (%) differed significantly according to a family history of cardiac disease: children with a positive family history had less negative strain values compared with those without ($p = 0.017$). No statistically significant subgroup differences were detected for strain rate or TAPSE ($p > 0.05$).

Table 4 Correlation of strain, strain rate, and TAPSE values with demographic, clinical and respiratory parameters in asthmatic children

	Strain (%)		Strain rate (s^{-1})		TAPSE (cm)	
	r	*p*	*r*	*p*	*r*	*p*
Age (year)	−0.146	0.228	−0.098	0.420	**0.478**	**<0.001**
BMI	0.102	0.401	−0.128	0.292	**0.398**	**0.001**
Asthma duration (year)	−0.076	0.531	0.029	0.810	**0.431**	**<0.001**
Time since last asthma attack (month)	−0.151	0.212	0.141	0.243	0.076	0.530
FEV1 (%)	0.079	0.514	−0.182	0.132	**0.301**	**0.011**
PEF (%)	−0.078	0.520	−0.161	0.182	0.200	0.097

Bold text indicates statistically significant results.
BMI, body mass index; FEV1, forced expiratory volume in 1 second; PEF, peak expiratory flow.

Within the asthma group, strain and strain rate were not associated with most clinical and laboratory variables, while TAPSE correlated positively with age, BMI, asthma duration, and FEV1. These relationships are biologically plausible given somatic growth effects on longitudinal annular excursion and the interplay between lung mechanics and RV loading conditions. However, correlation does not imply causation, and residual confounding (e.g., body size, conditioning, treatment intensity) may contribute.

An exploratory subgroup observation was that children with a family history of cardiac disease exhibited less negative RV strain values. Although this finding should be interpreted cautiously due to subgroup size and multiple comparisons, it may point to shared familial cardiovascular susceptibility or unmeasured exposures, and it suggests that family history could be considered when selecting patients for closer cardiopulmonary follow-up.

Clinically, our findings are relevant to asthma follow-up because they suggest that RV systolic mechanics may remain preserved in children with predominantly mild-to-moderate, stable asthma, while selected patients with greater asthma burden, poorer control, longer disease duration, or more intensive controller treatment may warrant closer cardiopulmonary attention. Targeted assessment of RV diastolic indices, such as tricuspid E′ and E/E′, may be useful when cardiac evaluation is clinically indicated.

This study has limitations. It was a single-center study with a modest sample size, a relatively small control group, and limited representation of severe asthma, limiting severity-stratified inference. The cross-sectional design does not evaluate longitudinal changes with control or treatment. The lack of systematic assessment of tricuspid regurgitation velocity and estimated pulmonary artery pressure limits the interpretation of RV afterload. Intra- and inter-observer reproducibility analyses for strain measurements were not conducted. Finally, strain values depend on vendor/software and pediatric norms, so comparisons across studies should remain platform-aware.

Conclusion

Children with predominantly mild-to-moderate, stable asthma demonstrated preserved RV systolic mechanics by 2D-STE and TAPSE, while tricuspid annular diastolic indices suggested subtle early RV diastolic involvement. These findings align with the broader literature indicating that diastolic abnormalities may precede deformation impairment and that strain differences are more prominent in severe or poorly controlled asthma.

Mandatory Disclosure on Use of Artificial Intelligence

The authors declare that no AI-assisted tools were used in the preparation of this manuscript. All references have been manually verified for accuracy and relevance.

Authors Contribution

ET, KN, NG, and ZUA conceptualized and supervised the study. ET, DO, NG, and ZUA provided patient care and collected clinical data. RO, KN, and MZB performed the cardiologic assessments and contributed to the acquisition of cardiac data. Statistical analyses were performed by ET and DO. ET, DO, and ZUA wrote the original draft of the manuscript. All authors reviewed and approved the final version of the manuscript.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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