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The evaluation of stiffness, distensibility, and strain of the abdominal aorta in asthmatic children

Astımlı çocuklarda aort sertliği, esnekliği ve geriliminin değerlendirilmesi

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ABSTRACT

Background: This study aims to investigate aortic stiffness, distensibility, and strain, which can be used to detect atherosclerosis in asthmatic children, and their impact on cardiac functions.

Methods: Between January 2012 and November 2014, a total of 21 pediatric patients (11 males, 10 females; mean age: 11.3±3.2 years; range, 6 to 15 years) with asthma and 17 healthy children (10 males, 7 females; mean age 12.8±3.8 years; range 7 to 16 years) were included. Using abdominal ultrasound, the stiffness, distensibility, and strain of the abdominal aorta were calculated. Echocardiographic examination was also performed on all children.

Results: Aortic stiffness was higher, while distensibility and strain values were lower in the asthmatic group, compared to the controls. Of the difference in the aortic strain, 30.3% was due to asthma, 22.5% to pulse pressure, 21.8% to mid-wall shortening fraction, and 17.2% to the left ventricular meridional wall stress. There was a very strong linear correlation between the left ventricular mass index and meridional wall stress (r=0.934), myocardial fiber stress (r=0.918), and predicted mid-wall fiber shortening for a measured fiber stress (r=0.918). Of the difference in the aortic distensibility, 40.6% was due to asthma, 18% to systolic blood pressure, and 12.2% to meridional end-systolic wall stress. Of the difference in the aortic stiffness, 24.7% was related to the diastolic blood pressure, 20.3% to ejection time, and 17.4% to the age variability.

Conclusion: According to our study results, aortic distensibility and strain decrease, while aortic stiffness increases in asthmatic children. Therefore, we suggest that asthmatic children should be followed closely for the development of atherosclerosis.

Keywords: Aorta; asthma; atherosclerosis; children; cardiac function.

ÖZ

Amaç: Bu çalışmada astımlı çocuklarda aterosklerozun tespitinde kullanılabilen aort sertliği, esnekliği ve gerilimi ve bunların kalp fonksiyonları üzerindeki etkisi araştırıldı.

Çalışma planı: Ocak 2012 - Kasım 2014 tarihleri arasında astımlı toplam 21 çocuk hasta (11 erkek, 10 kız; ort. yaş 11.3±3.2 yıl; dağılım 6-15 yıl) ve 17 sağlıklı çocuk (10 erkek, 7 kız; ort. yaş 12.8±3.8 yıl; dağılım 7-16 yıl) çalışmaya alındı. Abdominal ultrasonografi ile abdominal aortun sertliği, esnekliği ve gerilimi hesaplandı. Tüm çocuklarda ekokardiyografi çekildi.

Bulgular: Kontrollere kıyasla, astımlı grupta aort sertliği daha yüksek iken, esneklik ve gerilim değerleri daha düşüktü. Aort gerilimindeki farklılığın %30.3'ü astım, %22.5'i nabız basıncı, %21.8'i orta duvar kısalma fraksiyonu ve %17.2'si sol ventriküler meridyonel duvar stresi ile ilişkiliydi. Sol ventrikül kütle indeksi ile meridyonel duvar stresi (r=0.934), miyokardiyal fiber stresi (r=0.918) ve ölçülen fiber stresi için tahmini orta duvar fiber kısalması (r=0.918) arasında güçlü ve doğrusal bir ilişki bulundu. Aort esnekliğindeki farklılığın %40.6'sı astım, %18'i sistolik kan basıncı ve %12.2'si meridyonel sistol sonu duvar stresi ile ilişkiliydi. Aort sertliğindeki farklılığın %24.7'si diyastolik kan basıncı, %20.3'ü ejeksiyon zamanı ve %17.4'ü yaş değişkeni ile ilişkili bulundu.

Sonuç: Çalışma sonuçlarımıza göre, astımlı çocuklarda aort esnekliği ve gerilimi azalırken, aort sertliği artmaktadır. Bu nedenle, astımlı çocukların ateroskleroz gelişimi açısından yakından takip edilmesini önermekteyiz.

Anahtar sözcükler: Aort; astım; ateroskleroz; çocuk; kalp fonksiyonu.

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Asthma is a chronic inflammatory disorder of the airways which is associated with airway obstruction and hyperresponsiveness, and is characterized with recurrent episodes of wheezing, shortness of breath, and coughing.[1] Bronchial asthma affects several organs including the heart.[2] Atherosclerosis and asthma are both chronic inflammatory disorders. Asthma is not only associated with multiple markers of chronic systemic inflammation, but also with an increased risk of atherogenesis.[2] Chronic inflammation via common inflammatory pathways[3] is associated with atherosclerosis,[4] endothelial dysfunction,[5] and arterial stiffness[6] and adverse cardiovascular events, eventually.[7] In the literature, there are some studies investigating the relationship between the peripheral arterial stiffness and atherosclerosis and adverse cardiovascular outcomes.[8] Inflammation causes impairment of endothelial cell function and accelerates atherosclerosis.[5] Several studies reported that patients with asthma are faced with an increased risk of pulmonary embolism, hypertension, coronary heart disease, and heart failure.[3,7,8]

Elevated arterial stiffness is associated with myocardial infarction, heart failure, renal disease, stroke, and increased total mortality rates in adults.[9] Therefore, elevated arterial stiffness is considered to be a marker of subclinical atherosclerosis.[9] Arterial stiffness is a mechanical feature related to the vascular impedance and the afterload on the left ventricle (LV). Reduction in arterial distensibility leads to an increased pulse pressure and impedance of arterial flow, and pulsatile cardiac workload.[10]

In this study, we investigated aortic stiffness, distensibility, and strain, which can be used to detect atherosclerosis in asthmatic children, and its impact on cardiac functions.

PATIENTS AND METHODS

This retrospective study included a total of 21 pediatric patients (11 boys, and 10 girls; mean age 11.3±3.2 years; range, 6 to 15 years) who were randomly selected from the patient population with bronchial asthma and 17 healthy subjects (10 boys and 7 girls; mean age 12.8±3.8 years; range 7 to 16) years). Bronchial asthma was defined according to the Global Initiative for Asthma (GINA) criteria.[1] Exclusion criteria were as follows: existing comorbidities, upper or lower respiratory infection, allergic rhinitis, gastroesophageal reflux, or obesity; chronic cardiovascular or pulmonary diseases; acute asthma attack, or use of oral or inhaled steroids within the past four weeks.

The control group was selected from healthy children.

The study protocol was approved by the Bozok University Medical Faculty Ethics Committee. The study was conducted in accordance with the principles of the Declaration of Helsinki.

All children included in the study underwent a full history-taking and complete physical examination performed by a single physician. The heart rate and blood pressure (BP) of all children were recorded which were performed after 15 minutes of rest. The right brachial artery pressure was measured by a sphygmomanometer with an appropriate cuff. Both systolic (Ps) and diastolic (Pd) blood pressures were measured, and the mean value was recorded following three consecutive measurements. Pulse pressure (PP) was also calculated as PP = Ps-Pd.

Blood samples were obtained from the patients after a 12-hour fasting and were measured for glucose, total cholesterol, triglyceride, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) cholesterol.

Abdominal aorta artery was measured from the infrarenal segment, 2 cm distal to the renal arteries and at the widest arterial diameter at systole and the narrowest arterial diameter at diastole. The investigator performed and recorded three simultaneous arterial measurements in all children, using GE Logiq 7S Duplex ultrasonography (General Electric, Wauwatosa, WI, USA) with probe at a frequency of 3.1-10 MHz for B scan.

Aortic distensibility, strain, and stiffness were calculated as follows:

Distensibility (cm^2. dyn-1)= 2 x (arterial diameter systolic-arterial diameter-diastolic)/(arterial diameter-diastolic x pulse pressure).[10]

Strain= (systolic diameter-diastolic diameter)/ diastolic diameter).[10]

Stiffness (mmHg)= Logarithm (systolic BP/diastolic BP)/strain.[11]

An electrocardiogram was recorded for all patients. Transthoracic echocardiography was performed by a single experienced pediatric cardiologist who was blinded to the study groups. The following parameters were monitored echocardiographically: left ventricle end-diastolic pressure (LVEDP), left ventricle mass (LVM, g) according to the formula of Devereux,[12] peak early diastolic flow velocity (Peak E, cm/s) and peak late diastolic flow velocity (Peak A, cm/s), ratio between heights of early and late diastolic flow velocity peaks (E/A ratio) for mitral valve, deceleration time (DT, ms), LV meridional end-systolic wall stress (ESWSm, g/cm^2), mid-wall shortening

fraction (SFmid), heart rate corrected circumferential fiber shortening (VCFc), mid-wall VCFc, myocardial fiber stress (MFS, g/cm^2), and meridional LV wall stress (WSM, dyn/cm^2).

The following parameters were monitored by tissue Doppler echocardiography (TDE): annular peak velocity during late diastole (A'), annular peak velocity during early diastole (E'), isovolumetric relaxation time (IVRT), isovolumetric contraction time (IVCT), annular peak velocity during systole (S'), and ejection time (ET).

Mitral valve filling velocities were recorded from the apical four-chamber view with the pulse-wave Doppler during diastole. E, A, and DT were used as both ventricular diastolic function parameters. The ratios of E to A were calculated.

VCFc (circ/s)= (SF x (1500/heart rate)$^{0.5}$ / LV ET)

Midwall VCFc was calculated as= 0.0007 x fiber stress + 0.65

ESWSm was calculated by the method of Grossman et al.[13] and MFS according to the formula recommended by Regen.[14]

SFmid= [(LVED+h_d/2+s_d/2)-LVES-*mwst*]/(LVED + h_d/2 + s_d/2)

The *mwst* was calculated as= [(LVED + (h_d + s_d)/2]3 - LVED3 + LVES3)$^{0.333}$ –LVES]

s_d= end-diastolic septal thickness, h_d= left ventricular end-diastolic posterior wall thickness

Peak systolic (S') and early and late diastolic velocities (E' and A') were measured from the apical four-chamber view with the pulsed-wave Doppler sample volume at the mitral annulus.

Cardiac time intervals, including IVCT from the end of mitral flow to the beginning of aortic flow, IVRT from the end of aortic flow to the beginning of mitral flow, and ET from the beginning to the end of the mitral flow were also measured.

Statistical analysis

Statistical analysis was performed using SPSS version 16.0 software (SPSS Inc., Chicago, IL, USA). The Student's t-test, correlation analysis, regression, analysis, and the analysis of covariance (ANCOVA) were used to analyze data. The comparison of the arithmetic means of the radiographic results of aortic distensibility, strain, and stiffness between children with or without asthma was performed using independent t-test. The correlation between cardiac parameters and the dependent variables of aortic distensibility, strain, and stiffness were examined separately in patients and control subjects. The cardiac variables which were found to be significant in the correlation analysis were included into the ANCOVA as covariates to examine the differences in aortic distensibility, strain, and stiffness between the patient and control groups (fixed factor). In case of a highly significant correlation ($r \geq 0.80$) between the cardiac parameters, the variable with the highest degree of correlation with the aorta was included in the ANCOVA. Since the cardiac parameters can be affected by age, the age variable was also included in multiple ANCOVA as a covariate. Further tests were performed to determine whether these differences were due to asthma or other cardiac parameters. Prior to ANCOVA, the homogeneity of the group variances was assessed using the Levene's test and the test was performed, when homogeneity was ascertained. A p value of <0.05 was considered statistically significant.

RESULTS

There was no significant difference in age, systolic BP, pulse pressure, heart rate, fasting glucose, and HDL and LDL cholesterol levels between the groups (Table 1).

On the other hand, the aortic strain and distensibility were lower, while stiffness was higher in asthmatic children compared to the controls (Table 2).

There was also a significant correlation between the cardiac parameters and aortic distensibility, strain, and stiffness (Table 3). Aortic distensibility was positively correlated with S/E' and SFmid in the asthmatic group and positively correlated with systolic BP, pulse pressure, VCFc, SFmid and negatively correlated with IVCT, ET, ESWSm and age in the control group. Aortic strain was also positively correlated with SFmid in asthmatic children and positively correlated with LVM, WSM, MFSm, and mid-wall VCFc and negatively correlated with pulse pressure and ESWSm in the control group. Aortic stiffness was positively correlated with pulse pressure and ET and negatively correlated with diastolic BPd in the asthmatic group and positively correlated with ESWSm and negatively correlated with diastolic BP in the control group.

A total of 52.9% (R2=0.529) of the low distensibility in the aorta was specifically attributed to asthma, followed by systolic BP and ESWSm. Of this difference, 40.6% was due to asthma, 18% to systolic BP, and 12.2% to ESWSm (Table 4).

Of the low strain in aorta, 61% (R2=0.61) was specifically attributed to asthma, followed by pulse

Table 1. Demographic and clinical characteristics of asthmatic children and healthy controls

	Patients (n=21)		Control group (n=17)		
	n	Mean±SD	n	Mean±SD	*p**
Gender					
Male	11		10		
Female	10		7		
Age (years)		11.3±3.2		12.8±3.8	0.180
Systolic blood pressure (mmHg)		104.6±5.3		106.8±7.7	0.125
Diastolic blood pressure (mmHg)		60.1±6.2		62.5±5.0	0.132
Pulse pressure		38.3±5.3		41.3±6.2	0.140
Heart rate (bpm)		86.7±10.9		84.4±14.9	0.417
Glucose (mg/dL)		85.2±7.1		87.5±8.1	0.120
Total cholesterol (mg/dL)		147.5±22.6		152.5±23.6	0.122
Low-density lipoprotein cholesterol (mg/dL)		72.2±10.8		75.2±12.1	0.165
High-density lipoprotein cholesterol (mg/dL)		50.6±11.3		52.5±11	0.148
Triglyceride (mg/dL)		80.8±22		84±25	0.152

SD: Standard deviation; * Statistically significant (p<0.05).

pressure, SFmid, and WSM. Of this difference, 30.3% was due to asthma, 22.5% to pulse pressure, 21.8% to SFmid, and 17.2% to WSM. There was a very strong linear correlation between LVM with WSM (r=0.934), MFS (r=0.918) and mid-wall VCFc (r=0.918); as a result, changes in these parameters also affected the aortic strain (Table 5).

A total of 68.7% (R2=0.687) of the increased stiffness in the aorta was mainly attributed to diastolic BP, followed by ET and age. Of these differences, 24.7% was related to the diastolic BP, 20.3% to ET, and 17.4% to age variability. There was no significant correlation between asthma (10.8%) and ESWSm (9.7%) (values were within the reference ranges) (Table 6).

DISCUSSION

The present study investigated the elasticity properties of the abdominal aorta in children with asthma. On the basis of the association between chronic inflammation and atherosclerosis, we hypothesized that the impaired elasticity in children with asthma could possibly lead to an increased risk of atherosclerotic disease. Therefore, the abdominal aorta was assessed. During the atherosclerotic process, increased arterial stiffness and decreased arterial distensibility and strain have been previously reported.[15] Consistent with the previous findings, our results showed decreased distensibility and strain in the aorta with increased stiffness.

On the other hand, there is a scarcity of published data on the association between the childhood-onset asthma and atherosclerosis and only few studies evaluated elasticity in asthmatic children to date. Steinmann et al.[16] showed an increased arterial stiffness in children with asthma using carotid-femoral pulse wave velocity measurements. Weiler et al.[17] examined the arterial stiffness in peripheral large and small arteries and found no difference between the asthmatic adults and controls. These authors also reported a positive correlation between the small

Table 2. Radiographic measurements of aorta in asthmatic children and healthy controls

Radiological measurements	Group	n	Mean±SD	t*	Significant
Aortic distensibility	Asthma	21	11.7±6.1	2.120	0.043
	Control	17	17.3±9.3		
Aortic strain	Asthma	21	0.04± 0.02	4.260	<0.001
	Control	17	0.08± 0.03		
Aortic stiffness					
	Asthma	21	19.0±14.9	3.182	0.004
	Control	17	8.0±4.9		

SD: Standard deviation; * Independent t-test.

Table 3. Correlation between radiographic measurements of the aorta and cardiac parameters in asthmatic children and healthy controls

Cardiac variables	Aortic distensibility		Aortic strain		Aortic stiffness	
	Asthma	Control	Asthma	Control	Asthma	Control
Systolic blood pressure	0.299	0.597**	0.015	-0.171	-0.140	0.067
Diastolic blood pressure	0.111	0.235	0.285	0.457	-0.605**	-0.559*
Pulse pressure	0.207	0.490*	-0.313	-0.487*	0.543**	0.442
E/A	-0.245	-0.339	-0.213	-0.080	0.328	0.301
E'/A'	-0.204	0.112	-0.146	-0.030	0.242	0.127
S'/S	-0.377	0.076	-0.116	0.248	0.209	-0.283
IVCT	0.027	-0.547*	0.194	-0.175	-0.070	0.375
IVRT	0.017	-0.242	0.002	-0.007	0.022	0.150
ET	-0.201	-0.480*	-0.393	-0.420	0.631**	0.441
DT	0.119	-0.436	0.047	-0.128	-0.023	0.187
LVM	0.227	-0.212	0.175	0.590*	-0.162	-0.453
LVEDP	0.014	-0.101	0.021	0.136	-0.034	0.033
WSM	-0.237	-0.245	-0.142	0.536*	0.028	-0.361
ESWSm	-0.192	-0.611**	-0.053	-0.484*	0.122	0.604**
VCFc	0.276	0.611**	0.298	0.063	-0.279	-0.200
SFmid	0.519*	0.634**	0.462*	-0.026	-0.338	-0.151
MFS	-0.122	-0.114	-0.155	0.598**	-0.086	-0.424
Midwall VCFc	-0.122	-0.114	-0.155	0.598**	-0.086	-0.424
Age (years)	0.139	-0.556*	0.405	0.008	0.366	0.187

Pearson correlation, * Significant correlation at 0.05 (two-tailed). ** Significant correlation at 0.01 (two-tailed); E: Peak velocity during early diastole; A: Peak velocity during late diastole; E': Annular peak velocity during early diastole; A': Annular peak velocity during late diastole; S': Annular peak velocity during systole; IVCT: Isovolumetric contraction time; IVRT: Isovolumetric relaxation time; ET: Ejection time; DT: Deceleration time; LVM: Left ventricular mass; LVEDP: Left ventricle end-diastolic pressure; WSM: Meridional left ventricular wall stress; ESWSm: Meridional end-systolic wall stress; VCFc: Rate-corrected velocity of circumferential fiber shortening; SFmid: Midwall shortening fraction; MFS: Myocardial fiber stress; Midwall-VCFc: Predicted midwall fiber shortening for a measured fiber stress.

arterial elasticity index and forced expiratory volume at one second (FEV_1). Brachial-ankle pulse wave velocity measurements were performed to assess the arterial stiffness in the study by Sun et al.,[18] where an increased arterial stiffness was found among the adult asthmatic patients with stable disease, compared to the healthy controls. In the aforementioned study, a negative correlation between the brachial-ankle pulse wave velocity and FEV_1 was found. On the other hand, in a recent study by Ülger et al.,[15] no difference between the asthmatic children and control subjects was found in terms of the aortic stiffness parameters. Inhaled steroids were reported as a possible reason for decreased aortic stiffness. Ayer et al.[19] suggested

Table 4. Aortic distensibility by ANCOVA according to covariate variables

	Type 3 sum of squares	Df	Mean square	F	Significant	Partial eta squared
Corrected model	1279.102*	5	255.820	7.183	0.000	0.529
Intercept	6.077	1	6.077	0.171	0.682	0.005
Sistolic blood pressure	249.345	1	249.345	7.001	0.013	0.180
S'/S	41.312	1	41.312	1.160	0.290	0.035
ESWSm	157.729	1	157.729	4.429	0.043	0.122
SFmid	60.708	1	60.708	1.705	0.201	0.051
Group	777.580	1	777.580	21.833	0.000	0.406
Error	1139.692	32	35.615			
Total	10108.178	38				
Corrected total	2418.793	37				

Df: Degree of freedom; F: F-test; * R Squared= 0.529 (adjusted R squared= 0.455); S': Annular peak velocity during systole; ESWSm: Meridional end-systolic wall stress; SFmid: Midwall shortening fraction.

Table 5. Aortic strain by ANCOVA according to covariate variables

	Type 3 sum of squares	Df	Mean square	F	Significant	Partial eta squared
Corrected model	0.024*	5	0.005	9.992	0.000	0.610
Intercept	0.009	1	0.009	18.627	0.000	0.368
Age	0.000	1	0.000	0.297	0.590	0.009
Pulse pressure	0.004	1	0.004	9.316	0.005	0.225
SFmid	0.004	1	0.004	8.945	0.005	0.218
WSM a	0.003	1	0.003	6.658	0.015	0.172
Group	0.007	1	0.007	13.882	0.001	0.303
Error	0.015	32	0.000			
Total	0.152	38				
Corrected total	0.039	37				

Df: Degree of freedom; F: F-test; * R Squared= 0.610 (adjusted R squared= 0.549); SFmid: Midwall shortening fraction; WSM: Meridional left ventricular wall stress; * Since WSM was found to exhibit a highly significant linear correlation with the left ventricular mass (r=0.934), fiber stress (r=0.918) and predicted midwall fiber shortening for a measured fiber stress.(Midwall VCFc) (r=0.918), only WSM was included in the analysis.

that the reduction in the lung volume during early childhood might be associated with increased arterial stiffness. However, Bhatt et al.[20] reported no significant difference between the systemic inflammation markers and arterial stiffness in patients with chronic obstructive pulmonary disease.

Decreased arterial distensibility is a risk factor for cardiovascular disease. Several studies have shown the utility of aortic distensibility as a non-invasive method in the detection of early atherosclerosis among adults.[21] It has been also demonstrated that arterial distensibility decreases in several diseases, such as polyarteritis nodosa,[21] systemic lupus erythematosus,[22] and hypertension.[23] Mikola et al.[24] studied the aorta and carotid arteries in children and reported that aortic and carotid distensibility decreased with age, which was more pronounced in boys than in girls. Increased stiffness leads to decreased diastolic BP and increased pulse pressure, causing increased left ventricular afterload, and a wear-and-tear effect on the arterial wall tissue.[24]

Furthermore, ESWSm is an index of total forces per unit of myocardium.[25] It has been used as a measurement tool of myocardial afterload, the counter force limiting LV ejection.[25] Chamber geometry of the cardiac structure also has an effect on both ventricular contractility and myocardial performance and needs to be identified by measuring ESWSm and MFS. The ESWSm seems to be related to chamber shape and mass/volume ratio and displays the forces opposing predominantly meridional and circumferential planes. In the present study, we found that asthma disease, systolic BP, and ESWSm all had an effect on the aortic distensibility. We also observed a positive correlation between the aortic distensibility and systolic BP, and a negative correlation between the aortic distensibility and ESWSm.

Table 6. Aortic stiffness by ANCOVA according to covariate variables

	Type 3 sum of squares	Df	Mean square	F	Significant	Partial eta squared
Corrected model	4084.698a	6	680.783	11.347	0.000	0.687
Intercept	34.498	1	34.498	0.575	0.454	0.018
Age	392.600	1	392.600	6.544	0.016	0.174
Diastolic blood pressure	609.803	1	609.803	10.164	0.003	0.247
Pulse pressure	126.969	1	126.969	2.116	0.156	0.064
ET	475.020	1	475.020	7.917	0.008	0.203
ESWSm	200.059	1	200.059	3.335	0.077	0.097
Group	225.470	1	225.470	3.758	0.062	0.108
Error	1859.884	31	59.996			
Total	13466.404	38				
Corrected total	5944.582	37				

Df: Degree of freedom; F: F-test; * R Squared= 0.687 (adjusted R squared= 0.627); ET: Ejection time; ESWSm: Meridional end-systolic wall stress.

This study also showed that asthma disease, WSM, pulse pressure, LVM, SFmid, MFS and mid-wall VCFc affected the aortic strain. There was a positive correlation between LVM and SFmid and aortic strain and a negative correlation between WSM, MFS and mid-wall VCFc and aortic strain. We also observed decreased aortic distensibility and increased stiffness in the patients with asthma. On one hand, this leads to an increased LVM with an increased afterload. On the other hand, it increases the workload and stress on both meridional and circumferential fibers and also the myocardial stress. The ventricular contractility and myocardial performance may be affected by the chamber geometry, which should be identified by measuring ESWSm, mid-wall VCFc, and MFS. The latter, as the representative of myofiber afterload, is a more accurate index of the afterload for the hypertrophic or dilated LV.[26] As being systolic ejection index of deeper layers of myocardium, SFmid provides more physiologically appropriate measurements of LV in wall thickness and conditions such as LV concentric hypertrophy and provides information to assess the myocardial performance.[27]

Aortic stiffness was found to be related to the diastolic BP and ET. There was a negative relation with diastolic BP and a positive relation with ET and aortic stiffness. Increased stiffness caused prolonged ET with an increased afterload. There was no significant correlation between asthma and ESWSm (values were within reference ranges). In previous studies, ventricular mass and function have been shown to be associated with aortic stiffness.[28,29] To date, several studies have not shown a supporting finding for such a relationship.[29-31] However, in these studies, the systolic function of the heart was evaluated, but not the diastolic function. The most important factor in the development of cardiac hypertrophy is the end-systolic stress.[13] End-systolic stress is influenced by ventricular geometry, as well as the aortic function.[27,28] To overcome the end-systolic stress, there are some structural changes in the myocardium, which may result in myocardial systolic and diastolic stiffness, eventually.[32]

In the present study, there was no correlation between the aortic elasticity parameters and ventricular diastolic functions, such as E/A, IVRT, IVCT, DT.

To the best of our knowledge, we were unable to find any study evaluating the correlation between the aortic distensibility, strain, and stiffness with cardiac parameters, such as SFmid, WSM, MFS, mid-wall VCFc, or ESWSm in asthmatic children. Therefore, we were unable to compare our results with previous studies in the pediatric age group. We, hence, recommend further large-scale studies to confirm these findings.

In conclusion, aortic distensibility and strain decrease and stiffness increases in asthmatic children. Therefore, these individuals should be followed closely for the development of atherosclerosis.

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