



Elasticity and Lipids Changes in Children with Type I Diabetes Mellitus Compared with Controls and the Effect of Lipids on Elasticity in Diabetic Children

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Abstract

Background: Increased atrial elasticity is a marker of cardiovascular events. This study aimed to compare the parameters of elasticity and lipids in children with type 1 diabetes mellitus (T1DM) compared with controls and the effect of lipids on elasticity in children with diabetes.

Method: This case-control study was performed in 186 children aged 6 to 18 years. The aortic diameter was obtained from 3 cm above the aortic valve using M mode and was calculated as the distance between the medial edge of the anterior and posterior walls of the aorta at systole and diastole. AoS and AoD were recorded when the aortic wall was fully open. The parameters of aortic elasticity, aortic stiffness beta index, aortic tension, and elastic modulus of pressure were measured. Cholesterol, triglycerides, low-density lipoprotein, and high-density lipoprotein were measured. For data analysis, SPSS 20 considers the applicable error to be 0.05.

Results: The participants were matched by sex and age. The right CHO, LDL, HDL, and MPI lipids, systolic, diastolic, and aortic systolic blood pressure were different among patients. The analysis also showed that ASβI (MWU = 1582.50, $p < 0.001$) and PSEM (MWU = 1381.00 and $p < 0.001$) were higher when AS (MWU = 1204 and $p < 0.001$) and AoD (MWU = 1672.00 and $p < 0.001$) and AoD (MWU = 1672.00 and $p < 0.001$) were lower in patients than in controls. No lipid profiles were significantly correlated with stiffness parameters before and after controlling for age.

Conclusion: It was concluded that lipid profiles were different, and ASβI and PSEM were lower when AoS and AoD were higher in children with diabetes. None of the lipid profiles were significantly correlated with stiffness parameters before and after controlling for age.

Keywords

Aortic Elasticity, lipid, diabetes Type I, children

Introduction

Cardiovascular disease (CVD) is one of the leading causes of death worldwide, especially in Asia [1]. It is necessary to identify CVD risk factors to prevent this disease. In adults, some of these factors are obesity or obesity-related diabetes, but in pediatrics the risks are not well characterized [2]. Traditional risk factors such as age, gender, family history, and many modifiable factors such as hyperlipidemia, smoking, arterial injury, hypertension, and hyperglycemia predict CVD [3]. In addition, diseases such as thalassemia [4], celiac [5], obesity [6], and diabetes [7] are risk factors for CVD. Recently, arterial stiffness (AS) was introduced as a risk factor to predict CVD independent of the traditional risk factors mentioned above. Increased AS has also been shown to predict cardiovascular events in asymptomatic individuals without overt CVD [2,8]. AS is primarily used in research protocols and is not yet commonly used as a prognostic indicator in clinical practice [8]. Type I diabetes mellitus (T1DM) is characterized by a defect in insulin production and is the predominant form of diabetes in children and adolescents, probably

due to early childhood dietary patterns, infections, and obesity [9]. Serum lipid abnormalities represent an independent risk for CVD [10]. Among the lipids profiled, for instance, serum high-density lipoprotein (HDL) has been identified as having a protective effect on arteriosclerosis in middle-aged and elderly populations [3]. Several studies have examined the relationship between arteriosclerosis and other single atherosclerotic lipids, but have not only resulted in no confirmation but have also created new questions [11]. Low-density lipoprotein (LDL) was found to be independently associated with AS, and except for HDL, no other lipid profiled as a predictor for LDL was found to be a superior tool for identifying AS [12]. In this regard, the ratio of TG/HDL was found to be an independent determinant for AS in adolescents and young adults [11]. In children with T1DM, although controlling glycemia may not delay CVD chronic hyperglycemia, it could be induced dyslipidemia, endothelial dysfunction, arterial stiffness, autonomic neuropathy, left ventricle hypertrophy, and ventricular dysfunction that ultimately cause CVD [13,14] such as tissue Doppler imaging that is designed to characterize low-velocity, high-amplitude signals from myocardial motion for quantification of global and regional myocardial contractile and relaxing functions [15]. The main mechanism for age-related aortic stiffening is fracture and fragmentation of elastin fibers with repetitive stretch, leading to the transfer of stress to less extensible collagenous fibers in the arterial wall [16]. Considering the above-mentioned materials and facts, the present study aimed to investigate the effect of lipid profiles in

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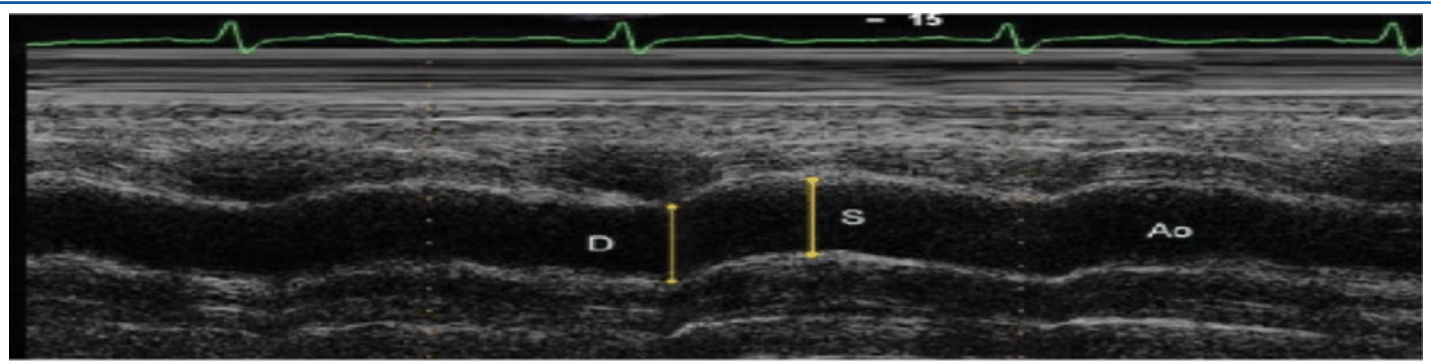


Figure 1 - Measurements of systolic (S) and diastolic (D) diameters of the ascending aorta are shown on the M mode tracing obtained at a level 3 cm above the aortic valve (4).

children with T1DM on arterial stiffing and the effect of some Doppler tissue-imaging parameters.

Methods

This case-controlled study was performed on 186 children aged 6 to 18 years with equal numbers of healthy and T1DM children in a pediatric cardiac center in collaboration with the center for specific diseases in Ali Asghar Hospital, Zahedan, Sistan, and Baluchestan province, Iran, for one year beginning on April 2020. Consent was obtained from the participants or their guardians after the study was approved (IR.ZAUMS.REC.1400.095).

1. Criteria

Diabetes was confirmed in participants with fasting blood glucose >125 or random blood glucose >200 mg/dl. Exclusion criteria were cardiac disease, such as ischemic, hypertensive disease, cardiomyopathy, valvular heart disease, congenital heart disease, myocarditis, and hypothyroidism, kidney, celiac, and thalassemia diseases in potential participants.

2. Echocardiography Measurements

Physical examination, chest X-ray, and echocardiography were performed on children using Mylab 60 with transducer 3, 8 (made in Italy), running three cycles and considering the average result. Left ventricular end diastolic dimension (LVDD), posterior wall dimension in diastole (PWD), interventricular septal dimension in diastole (IVSD), interventricular septal dimension in systole (IVSS), ejection fraction (EF), fractional shortening (FS), left ventricular mass (LVM), and left ventricular mass index (LVMI) were measured using conventional echocardiography of the left side and estimated from three cardiac cycles. LVMI was calculated using the following formula: $LVM (g) = 0.8(1.04(LVDD + PWD + IVSD)^3 - LVDD^3) + 0.6$. The sample volume was placed at the tips of the mitral and tricuspid valve leaflets in the apical four-chamber view to allow measurement of (a), which is the interval time between the end and the beginning of trans-mitral flow and trans-tricuspid. The sample volume was then

relocated to the left ventricular outflow tract just below the aortic valve (5-chamber apical view) to measure (b), which is the left ventricular ejection time. The right ventricular outflow velocity pattern was also recorded from the parasternal short axis view, using the measurement of the Doppler sample volume just distal to the pulmonary valve (b). Right ventricle and left ventricle myocardial performance index (MPI) was obtained by dividing the sum of the isovolumic relaxation time (IRT) and the isovolumetric contraction time (ICT) by the ejection time (ET) ($MPI = (ICT + IRT)/ET$) [7].

3. Aorta Parameters

After echocardiography, the aortic diameter was obtained from 3 cm above the aortic valve using the M mode. Aortic diameters were calculated as the distance between the anterior and posterior wall inner edges of the aorta at systole and diastole. AoS was recorded when the aortic wall was fully open. AoD was recorded simultaneously when the QRS peak was seen on electrocardiographic (ECG) recordings. Measurements were taken during three consecutive pulses and the mean was calculated. The ascending aortic diameters were recorded in M-mode approximately 3 cm above the aortic valve from parasternal long axis views. The systolic aortic diameter was measured at the time of maximum anterior motion of the aorta, whereas the diastolic diameter was measured at the start of the QRS complex in electrocardiography (Figure 1).

4. Blood Pressure

Blood pressures (BP) were measured from the brachial artery with a sphygmomanometer after at least 5 min resting in a supine position. Three measurements, at least 2 min apart, were applied, and the average of the closest two readings recorded. A pressure drop rate of approximately 2 mm Hg/s was applied, and Korotkoff phases I and V were used for systolic and diastolic BP, respectively.

5. Aortic Elasticity Parameters

Aortic elasticity parameters were calculated as follows:

- Aortic strain (%) = $(\text{aortic SD} - \text{aortic DD}) \times 100 / \text{aortic DD}$
- Aortic stiffness beta index = $\text{natural logarithm} (\text{systolic BP} / \text{diastolic BP}) / ([\text{aortic SD} - \text{aortic DD}] / \text{aortic DD})$

- Aortic distensibility ($\text{cm}^2 \cdot \text{dyne}^{-1} \cdot 10^{-6}$) = $2 \times ([\text{aortic SD} - \text{aortic DD}] / [\text{aortic DD}] / (\text{SBP} - \text{DBP}))$
- Pressure strain elastic modulus = $(\text{SBP} - \text{DBP}) / ([\text{aortic SD} - \text{aortic DD}] / [\text{aortic DD}])$ [5,6].

6. Lipid profiles

Lipid profiles of CHO mg/dl, HDL mg/dl, LDL mg/dl, and TG mg/dl were considered for the study with cut points of CHO >200 mg/dl, HDL <40 mg/dl, LDL >130 mg/dl, and TG >150 mg/dl as abnormal levels [7].

7. Anthropomorphic Measurements

Height and weight were measured by an experienced nurse with standard equipment. Height was measured in a standing position with a balance using a scaled ruler and weight measured using a RASA scale factor with an error of 100 g (made in Iran). BMI was calculated as $\text{weight}/\text{height}^2$ (kg/m^2).

8. Statistical Analysis

Data were analyzed using SPSS 20.0 (SPSS Inc., Chicago, IL, USA). After normality test, the *t*-test was used to compare mean values of normal quantitative variables, and the Mann-Whitney U test was used for the variables with skewed distribution. In correlation analyses, the Pearson chi-square test was used; *p* value ≤ 0.05 was considered to be the level of significance.

Results

In the present study, out of 186 participants, 53.2% were boys ($X^2 = 2.61$, $p = 0.106$), when this trend for patients and controls was 47.3% and 59.1%, respectively. After testing for normality, it was found that in all participants, FS, CHO, right MPI, and AoD have a normal distribution while in the patients, the variables of height, weight, EF, FS, left MPI, AoS and AoD have a normal distribution.

Comparison of variables in patients and controls showed similar age (MWU = 4166.00 and $p = 0.665$), and the variables of CHO ($t = 5.46$ and $p < 0.001$), LDL (MWU = 3179.00, and $p = 0.002$), HDL (MWU = 1524.00 and $p < 0.001$), right MPI ($t = 8.01$ and $p < 0.001$), SBP (MWU = 3268.00 and $p = 0.003$), DBP (MWU = 2897.00 and $p < 0.001$), and AoS (MWU = 2336.00 and $p < 0.001$) were statistically different. The analysis also showed that ASβI (MWU = 1582.50, $p < 0.001$) and PSEM (MWU = 1381.00 and $p < 0.001$) were higher when AoS (MWU = 1204 and $p < 0.001$), and AoD (MWU = 1672.00 and $p < 0.001$) were lower in patients than in controls (Table 1).

Figure 2 shows the results of the box plot presentation of cardiac sclerosis parameters, lipids, Doppler results, and systolic and diastolic aortic diameters in patients and controls. The statistics of minimum, Q1, median, Q3, and maximum have been presented in the figures.

Table 2 shows the correlation between stiffness parameters and other variables before and after controlling for age of patients.

Table 1 - Comparing Doppler tissue imaging, aortic stiffness, and lipid profile levels in children with diabetes and in controls

Variab.	Groups	Mean	SD	Mean rank	Test value	P value	Variab.s	Mean	SD	Mean rank	Test value	P value
Age	Case	10.84	3.43	95.2	4166	0.665	LDL	90.61	23.93	105.82	3179	0.002
	Control	10.8	2.85	91.8				78.55	21.48	81.18		
Height	Case	136.8	18.9	69.55	2097	<0.001	HDL	54.29	11.86	63.39	1524.5	<0.001
	Control	153.7	12.63	117.45				69.71	11.73	123.61		
Weight	Case	32.92	11.81	70.42	2178	0.001	SBP	97.8	10.39	82.14	3268	0.003
	Control	44.38	12.31	116.58				101.49	9.8	104.86		
EF	Case	75.55	5.93	84.02	3442.5	0.016	DBP	62.04	7.45	78.15	2897	<0.001
	Control	77.57	4.93	102.98				66.46	7.67	108.85		
FS	Case	43.96	5.4	83.22	-2.58	0.011	AOS	2.27	0.32	114.88	2336.5	<0.001
	Control	45.87	4.68	103.78				2.01	0.31	72.12		
LVM	Case	47.62	20.43	90.42	4038.500	0.436	AOD	1.87	0.29	96.6	4036.5	0.433
	Control	40.24	21.38	96.58				1.85	0.32	90.4		
Left MPI	Case	0.79	0.1	134.06	552	<0.001	ASβI	10.76	15.66	122.99	1582.00	<0.001
	Control	0.52	0.13	52.94				2.52	1.13	64.01		
Right MPI	Case	0.76	0.12	121.12	8.01	<0.001	AS	9.35	7.00	59.95	1204.00	<0.001
	Control	0.63	0.11	65.88				22.40	11.70	127.05		
TG	Case	124.52	76.17	100.3	3692	0.085	AD	0.005	0.006	64.98	1672.50	<0.001
	Control	94.39	30.07	86.7				0.014	0.008	122.02		
CHO	Case	155.54	37.52	112.67	5.46	<0.001	PSEM	8.80	13.24	125.15	1381.50	<0.001
	Control	125.78	36.83	74.33				1.96	0.90	61.85		

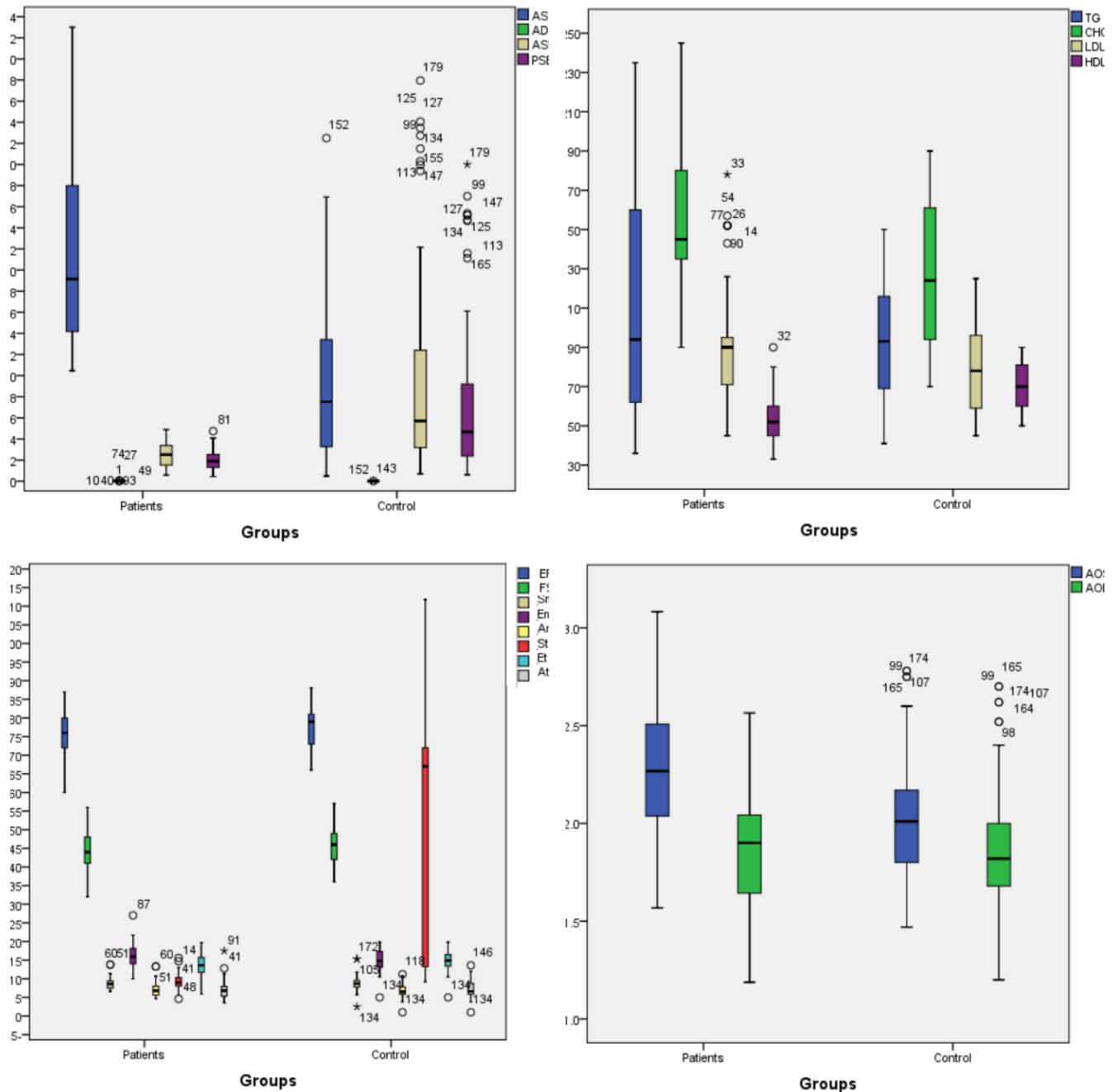


Figure 2 - Boxplot presentation of heart-stiffening parameters, lipids, Doppler findings, and diameter of aorta in systole and diastole in patients and in controls.

The analysis showed that before controlling for age, duration was positively and significantly correlated with AS β l ($r = 0.223$, $p = 0.032$) and PSEM ($r = 0.255$, $p = 0.014$). HbA1c has a positive and significant correlation with PAS ($r = 0.274$, $p = 0.008$) and DBP ($r = 0.265$, $p = 0.010$). The Doppler parameters of EF and FS were negatively and significantly correlated with SBP. On this point, correct MPI has a positive and statistically significant correlation with SBP ($r = 0.219$, $p = 0.035$) and HATTr ($r = 0.213$, $p = 0.040$). The results after testing show a small difference (visible in the table). None of the lipid profiles

were significantly correlated with stiffness parameters before and after controlling for age.

Discussion

Arterial stiffness parameters are associated with increased risk of cardiovascular events that are affected by diabetes and other diseases [17]. The results from the present study demonstrate that

Table 2 - Correlation between aortic stiffness parameters and Doppler tissue imaging, as well lipid profiles before and after controlling for age in patients.

		Before controlling for age							
Variables	Statistics	SBP	DBP	AOS	AOD	AS	AD	ASβI	PSEM
Duration	r	0.073	-0.002	0.085	0.087	-.160-	-.197-	0.223	0.255
	p	0.489	0.984	0.419	0.407	0.126	0.058	0.032	0.014
HbA1c	r	0.274	0.265	-.004-	-.091-	-.008-	0.031	-0.063	-0.058
	p	0.008	0.01	0.97	0.384	0.939	0.766	0.548	0.578
EF	r	-0.254	-0.224	-.056-	0.036	0.012	0.051	-.066-	-.110-
	p	0.014	0.031	0.597	0.735	0.909	0.625	0.532	0.295
FS	r	-0.241	-0.203	0.01	0.089	0.026	0.062	-.059-	-.098-
	p	0.02	0.051	0.924	0.394	0.802	0.557	0.574	0.35
Left MPI	r	0.148	0.186	-.014-	-.050-	0.158	0.177	-.059-	0.003
	p	0.156	0.074	0.897	0.635	0.131	0.09	0.576	0.978
Right MPI	r	0.219	0.213	-.057-	-.063-	-.053-	0.015	0.067	0.115
	p	0.035	0.04	0.589	0.547	0.616	0.886	0.525	0.273
LVM	r	.055	-.024	.047	.123	-.131	-.142	.108	.106
	p	.454	.748	.524	.093	.076	.053	.142	.151
TG	r	-.025-	-.077-	0.052	0.026	0.097	-.007-	0	-.013-
	p	0.809	0.464	0.62	0.801	0.357	0.951	0.998	0.902
CHO	r	-.050-	-.075-	-.080-	-.038-	-.159-	-.151-	0.072	0.112
	p	0.633	0.472	0.444	0.719	0.129	0.148	0.49	0.287
LDL	r	-.092-	-.060-	-.067-	-.030-	-.077-	-.053-	-.007-	-.005-
	p	0.378	0.569	0.523	0.776	0.466	0.612	0.946	0.963
HDL	r	-.067-	-.010-	0.078	0.091	-.107-	-.082-	0.115	0.109
	p	0.523	0.921	0.455	0.384	0.306	0.437	0.272	0.299
		After controlling for age							
duration	r	0.008	-0.048	-0.011	-0.01	-0.176	-0.221	0.219	0.25
	p	0.942	0.649	0.915	0.922	0.094	0.034	0.036	0.016
HbA1c	r	0.294	0.271	-0.018	-0.129	-0.009	0.029	-0.064	-0.059
	p	0.004	0.009	0.864	0.22	0.929	0.781	0.545	0.574
EF	r	-0.285	-0.236	-0.076	0.038	0.011	0.051	-0.066	-0.11
	p	0.006	0.024	0.469	0.719	0.915	0.632	0.531	0.295
FS	r	-0.284	-0.223	-0.016	0.084	0.023	0.057	-0.061	-0.1
	p	0.006	0.033	0.88	0.426	0.826	0.587	0.566	0.341
Left MPI	r	0.209	0.222	0.054	0.01	0.167	0.191	-0.055	0.008
	p	0.046	0.033	0.607	0.925	0.111	0.068	0.602	0.938
Right MPI	r	0.238	0.219	-0.078	-0.087	-0.054	0.014	0.066	0.115
	p	0.022	0.036	0.461	0.411	0.612	0.894	0.53	0.277
LVM	r	.047	-.029	.035	.127	-.130	-.139	.111	.108
	p	.528	.691	.640	.084	.078	.059	.133	.145
TG	r	-0.051	-0.094	0.028	-0.005	0.093	-0.013	-0.002	-0.016
	p	0.629	0.37	0.789	0.966	0.379	0.903	0.986	0.882
CHO	r	-0.012	-0.052	-0.031	0.024	-0.152	-0.141	0.077	0.117
	p	0.909	0.624	0.771	0.82	0.148	0.179	0.467	0.265
LDL	r	-0.019	-0.01	0.052	0.102	-0.063	-0.031	0.001	0.005
	p	0.861	0.924	0.621	0.331	0.553	0.767	0.996	0.96
HDL	r	-0.187	-0.081	-0.078	-0.065	-0.131	-0.115	0.108	0.099
	p	0.074	0.445	0.457	0.537	0.214	0.276	0.304	0.349

CHO, LDL, HDL, right MPI, SBP, DBP, AoS and AoD manifested differently in diabetic patients: AS β I and PSEM were higher, and AS and AD were lower in children with diabetes. Noori et al. [7] found that mean rank of left and right MPI were higher in children with diabetes (136.77 and 123.14) compared to the controls (56.23 and 69.86, respectively) ($p < 0.001$).

Acar et al. [18] found that left and right MPI values were higher in children with DMTI, and the right MPI values were not significant. Ozdemir et al. [19] reported that MPI varied between children with diabetes and healthy controls. AS parameters are recognized as surrogate endpoints to predict future cardiovascular events that are measured by noninvasive imaging modalities [20]. For example, Ayhan et al. [21] found that aortic strain ($8.0\% \pm 1.5\%$ vs. $13.1\% \pm 3.3\%$; $p < 0.001$) and AD ($3.6 \pm 1.1 \text{ cm}^2 \cdot \text{dyn}^{-1} \cdot 10^{-3}$ vs. $6.0 \pm 2.1 \text{ cm}^2 \cdot \text{dyn}^{-1} \cdot 10^{-3}$; $p < 0.001$) were significantly decreased in patients with diabetes compared to controls, respectively.

Duarte et al. [22] found that children with diabetes had a higher augmentation index and augmentation pressure compared to the control group. Shah et al. [20] assessed three measures of stiffening--PWV, AI75, and Brach D--in diabetes patients. They received higher PWV (5.9 ± 0.05 vs. 5.7 ± 0.1 , $p < 0.05$), higher AI75 (1.3 ± 0.6 vs. -1.9 ± 0.7 , $p < 0.05$), and lower Brach D (6.2 ± 0.1 vs. 6.5 ± 0.1 , $p < 0.05$) in diabetic patients compared to controls. Heier et al. [23] found, higher PWV in diabetic patients when AD was lower but the difference was not significant. Similarly, Llauredó et al. [24] found that diabetic patients had higher arterial stiffness (PWV) compared with healthy control subjects (men: 6.9 vs. 6.3, $p < 0.001$; women: 6.4 vs. 6.0, $p = 0.023$). They also showed that these changes remained significant after adjusting for cardiovascular risk factors. In a regression analysis, they showed that age was associated independently with PWV (AS β I = 0.550). Heier et al. [23] reported that PWV (4.10 ± 4.58 vs. 3.90 ± 4.04 , $p = 0.045$) was significantly higher in the diabetic patients compared with controls, and all lipid profiles were higher in diabetic children, when CHO and LDL were significant. Mostofizadeh et al. [25] reported that lipid profiles were high in Iranian children with T1DM. The most common lipid profiles abnormality in these children was hypercholesterolemia and then LDL. Kim et al. [26], Zabeen et al. [27] and Mostofizadeh et al. [25] reported that lipid profiles changed in diabetic children. The present study demonstrated that except for TG, the other lipids, CHO and LDL, were higher when HDL was lower in diabetic children. The high level of CHO, triglyceride, LDL, and low HDL may be due to obesity, increased calorie intake, and lack of muscular exercise in these patients. The estimation of lipid peroxide along with other lipid profiles in diabetes is very useful as it may serve as a practical way to monitor the prognosis of the disease. The detection of risk factors in the early stage of the disease will help the patient to improve and reduce the morbidity rate [7]. Urbina et al. [28] conducted a study on subjects aged 10 to 26 years old to assess the effect of TG/HDL on arterial stiffness. They classified patients into three groups of low, middle, and high TG/HDL. The result was that when TG/HDL increased, PWV increased and BrachD decreased. Wang et al. [29] found that TC, TG, and LDL levels were positively correlated with

arterial stiffness (PWV), and HDL was negatively correlated with PWV. After controlling for age, the result was that TG and HDL were associated with arterial stiffness. Wang et al. [30] reported that those with higher aortic stiffness had higher cholesterol, triglyceride, and low-density lipoprotein cholesterol levels and lower high-density lipoprotein cholesterol levels. In a recent study, Noori et al. [7] showed that Doppler tissue imaging parameters of left A/A' had a significantly ($p = 0.016$) higher value in diabetic patients with normal TG (8.22 ± 2.12) compared with those patients with abnormal TG (7.13 ± 1.68). In the present study, we found that cholesterol had stronger effects on Doppler findings in the T1DM children. An increased lipids level may be due to the abnormality in lipid metabolism in the population [27] when in diabetes mellitus, elevated levels of lipid peroxide may be due to the alteration of the erythrocytes' membrane function. This cause inhibits the activity of superoxide dismutase enzyme leading to accumulation of superoxide radicals which cause the maximum lipid peroxidation and tissue damage in diabetes [30, 31]. Consequently, there is a clear association between lipid peroxide and glucose concentration, which may also play a role in increased lipid peroxidation in diabetes mellitus [31]. Ayhan et al. [21] found that the serum lipids were associated with CVDs but the correlation with AS was unclear. Zhao et al. [3] showed that only HDL was associated with a high AS, while Yiming et al. [32] reported that only TG levels were significantly correlated with AS. In this regard, Zhan et al. [33], found, in a large cross-sectional study, a correlation between AS parameters and all lipid variables in patients with hypertension. This correlation between the lipid profiles and PWV perhaps is due to excess lipid and cholesterol binding to the arterial intima and accumulating in the arterial wall, subsequently leading to AS [29]. Additionally, oxidative and nitrosative stress caused by excess lipids accelerates AS changes [34]. Shah et al. [20] in the presence of age, sex, race, adiposity, blood pressure, and lipids found a correlation between AS and heart function. After controlling these factors, heart function was still correlated with AS. It has been reported in the Wang et al. [29] study that cholesterol, TG, and LDL were positively correlated with AS and HDL was negatively correlated. After controlling for age, BMI, only TG levels were correlated with AS. After adjusting age, sex, BMI, and other cardiovascular risks, HDL was negatively correlated with stiffing aorta when TC and TG had a positive correlation, while no correlation was observed between LDL and aortic stiffing [35]. Although large AS increases with age independently of the presence of cardiovascular risk factors or other associated conditions, the extent of this increase may depend on several factors including environment, genetics, and diseases [25, 36]. Patients with heart failure, end stage renal disease, and those with atherosclerotic lesions often develop central artery stiffness. Decreased carotid distensibility, increased arterial thickness, and presence of calcifications and plaques often coexist in the same subject. However, relationships between these three alterations of the arterial wall remain to be explored [37]. Finally, lipids and AS may be associated with chronic inflammation in the vessel wall. Leukocytes stimulated by lipids release a variety of cytokines and adhesive molecules, which in turn leads to leukocytes adhering

to vascular endothelium and penetrating the intima, resulting in increased vascular resistance [38]. Small sample size was the only study limitation such that with this insufficient sample, it is hard to find the differences in the parameters or the association between the parameters of the study.

Conclusions

From this study, we concluded that ASβI and PSEM were higher when aortic strain and AD were lower in patients. The measures of HbA1c, EF, FS, left MPI, right MPI, and LMI had a significant correlation with at least one of the SBP, DBP, AOS, and AOD parameters before and after controlling for age. None of the lipid profiles showed significant correlation with stiffening parameters before and after controlling for age. Since the aortic stiffening was accelerated by diabetes, patients with diabetes have to control their diabetes with specific programs.

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Conflict of interest

The authors declare no conflict of interest.

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Authors' contributions

NMN: Concept and design, data acquisition, interpretation, final approval, and agree to be accountable for all aspects of the work. MNM: Data acquisition, interpretation, final approval and consent to be accountable for all aspects of the work. AT: Data analysis, Interpretation, drafting, final approval.

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