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Original Article

Assessment of aortic elasticity parameters in obese and overweight children

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ABSTRACT

Background. Aortic elasticity is a predictor and recognized factor for future cardiovascular events in children. The aim of the study was to evaluate the aortic stiffness in obese and overweight children compared to healthy ones.

Methods. The study evaluated 98 sex matched children aged 4 to 16 years that were equally distributed in asymptomatic obese or overweight and healthy children groups. All the participants were free of any heart diseases. Arterial stiffness indices were determined using two-dimensional echocardiography.

Results. The mean ages in the obese and healthy children were 10.40±2.50 years and 10.06±1.53 years, respectively. Aortic strain was significantly higher in obese children (20.70±5.04%), compared to healthy (7.06±3.77%) and overweight children (18.59±8.08%, p<0.001). Aortic distensibility (AD) was significantly higher in obese children (0.010±0.005 $cm^2dyn^{-1}x10^{-6}$), compared to healthy (0.0036±0.004 $cm^2dyn^{-1}x10^{-6}$) and overweight children (0.009±0.005 $cm^2dyn^{-1}x10^{-6}$, p<0.001). Aortic strain beta (ASβ) index, was significantly higher in healthy children (9.26±6.17). Pressure-strain elastic modulus (PSEM) was significantly higher in healthy children (7.52±4.76 kPa). Systolic blood pressure increased with body mass index (BMI) significantly (p<0.001) but diastolic blood pressure did not change (p=0.143). BMI had significant effect on arterial stiffness (AS) (β=0.732, p<0.001), AD (β=0.636, p<0.001), ASβ index (β=-0.573, p<0.001) and PSEM (β=-0.578, p<0.001). Age had significant effect on systolic diameter of the aorta (β=0.340, p<0.001) and diastolic diameter of the aorta (β=0.407, p<0.001).

Conclusions. We concluded that aortic strain and aortic distensibility increased in obese children when aortic strain beta index and PSEM decreased. This result suggests that, as atrial stiffness is a predictor for future heart diseases, dietary treatment for children with overweight or obese status is important.

Key words: aortic elasticity, obese, overweight, children.

Arterial stiffness (AS) is a new detectable manifestation of damaging structural and practical adjustments in the vessel wall. It is also often an effect of incorporating cardiovascular hazard elements of the blood vessel.[1,2] Estimating AS offers the possibility to detect the activities of coronary vascular disease (CVD) earlier than the primary signs, which is an essential component of health care.[2] An increase in AS takes place because of arteriosclerosis and aging that is probably tormented by numerous components of hereditary inclination and CVD danger elements which include weight problems or hypertension.[2-4] In childhood, most of the large arteries are very elastic, however stiffen with aging.[3-5] In addition, AS increases in many conditions which include celiac disease, asthma, diabetes mellitus, chronic kidney disease, thalassemia and obesity.[5-10] Obese children are at risk for many persistent diseases which include excessive blood pressure, CVD, dyslipidemia and diabetes and each one of these disorders may have a robust impact on AS.[11] In a study, it has been reported that the prevalence of obesity is 10.1% and 4.79% in children aged 6 and 18 years, respectively, with more

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occurrence in females; and in these children, approximately 70% have one and 39% have two or more risk factors for CVD.[12-14] In addition to association with heart disease in children, these CVD risk factors and obesity are also associated with an increased prevalence in adults.[15] Flynn et al.[16], found that there is a rising prevalence of childhood systolic and diastolic blood pressure in obese children. Ayer et al.[17], observed an increase in total cholesterol (CHO), low-density lipoprotein (LDL), and triglycerides (TG) and lower high-density lipoprotein (HDL) level in obese participants. In this regard, Ayer et al.[17], Dangardt et al.[18]▪found that AS increased rapidly in obese children; and Charakida et al.[19] found that vascular dysfunction was not observed among prepubertal obese children. AS is recognized as a marker of cardiovascular risk but it has also been reported that it is increased in obese children without overt cardiovascular diseases.[20] Therefore, the aim of this study was to evaluate the effect of obesity on AS in children and compare the results to children of normal weight.

Material and Methods

Study design

This case-control study involved 98 children and adolescents between the ages of 4 and 16 who were evenly divided into two groups: obese or overweight children and controls. Children with a normal BMI served as the controls. The research was carried out in 2020, at Ali Asghar Hospital in Zahedan City, Sistan and Baluchestan province, Iran, in collaboration with the center for specific diseases.

Criteria

For each participant, the following exclusion criteria were taken into consideration: diabetes mellitus, hypertension, dyslipidemia, systemic autoimmune disease, active infection, evidence of liver, renal, or lung disease, smoking exposure, concurrent treatment with antihypertensive medications, lipid-lowering medications, and a positive family history of dyslipidemia are all co-morbid conditions.

Echocardiography measurements

The medical history, physical examination, chest X-ray, and echocardiography were the primary procedures performed on patients. The same pediatric cardiologist used a MyLab 60 Class C, Esaote, with a transducer of 3 to 8 MHz to perform echocardiography on the participants. Measurement was repeated for three cycles and the average was taken into consideration in order to achieve high precision in the results of echocardiography. Participants underwent an echocardiogram without having to hold their breath. M-mode echocardiography revealed the following: diastolic and systolic diameters of the aorta, respectively (AOD and AOS). The M-mode was used to obtain ascending aorta from 3 centimeters above the aortic valve following a routine echocardiographic examination. The distance between the inner edges of the aorta's anterior and posterior walls at systole and diastole was used to calculate aortic diameters. The mean was determined by taking measurements during three consecutive pulses.[9]

Blood pressure measurements

A sphygmomanometer was used to measure blood pressure (BP) from the brachial artery at the level of the heart after at least 5 minutes of rest in the supine position. Three measurements were carried out, at least 2 min. apart, and the average of the two readings that were closest together was recorded. Korotkoff phases I and V were utilized for the systolic and diastolic BP levels, respectively, with a pressure drop rate of approximately 2 mm Hg/second. Pulse pressure (PP) was calculated by subtracting systolic BP from diastolic BP.

Assessment of aortic elasticity

The systolic and diastolic ascending aortic diameters were recorded in M-mode under echocardiographic and electrocardiographic guidance 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 while the diastolic diameter was measured at the start of the QRS complex in electrocardiography. The aortic elasticity of the aorta was evaluated using the formulas listed below.[9]

Aortic strain (%) = (aortic SD [systolic diameter] - aortic DD [diastolic diameter]) x100 / aortic DD.

Aortic stiffness beta (ASβ) index = natural logarithm (systolic BP / diastolic BP) / ([aortic SD-aortic DD] / aortic DD).

Aortic distensibility ($cm^2dyn^{-1}x10^{-6}$) = 2 x ([aortic SD - aortic DD] / aortic DD) / (systolic BP-diastolic BP).

Pressure strain elastic modulus (kPa) = (systolic BP–diastolic BP) / ([aortic SD–aortic DD] / aortic DD)

Anthropomorphic measurements

An experienced expert measured the participants' height and weight using standard equipment. Height was measured to the nearest 0.1 cm and weight to the nearest 0.1 kg, wearing light indoor clothes without shoes using an integrated calibrated weight and stadiometer (ADE, Modell MZ10023, Hamburg, Germany). BMI (kg/m^2) was calculated. Those with a BMI between 18-25 were assigned to the control group, those with a BMI between 25.0 to 30 were defined as overweight and those who had a BMI higher than 30 was defined as obese. The overweight and obese participants were assigned to the study group.

Ethical approval

The study, a project proposed to the Children and Adolescent Health Research Center, was approved as by the Ethics Committee of Zahedan University of Medical Sciences, Zahedan, Iran (IR.ZAUMS.REC.1400.095, 2021-5-26). Written informed consent form was obtained from the participants or their guardians.

Statistical analysis

The software of SPSS 20.0 was used to analyze the data (SPSS Inc., Chicago, IL, USA). Kolmogorov-Smirnov test was used to assess distribution of continuous variables where homogeneity was tested. Independent t-test was used to compare two mean values of quantitative variables with normal distribution while Mann-Whitney U test was used to compare quantitative variables with skewed distribution. One-way ANOVA test was used for comparison between three groups, followed by Tukey post-hoc test and Kruskal-Wallis test was used for comparison between three groups, followed by Dunn's post-hoc test. Linear regression was used to determine how certain variables affected the parameters of the heart's stiffness. A significance level of 0.05 was deemed statistically significant.

Results

The mean age of all participants was 10.23±2.07 years; such that in the overweight or obese group it was 10.40±2.50 years and it was 10.06±1.53 years in the healthy children. Of the participants, 41 (41.8%) were female and 57 (58.2%) were male. In the overweight or obese children group 18 (36.7%) were female and 31 (63.3%) were male, in the healthy children 23 (46.9%) were female and 26 (53.1%) were male (p=0.306).

All variables had had normal distribution, except AOS and AOD. Primary analysis showed that atrial strain, AD had increasing trends by increasing BMI when ASβ index and PSEM had declining trends. Atrial strain increased with BMI and was higher in obese children (20.70±5.04 $cm^2dyn^{-1}x10^{-6}$), compared to normal (7.06±3.77 $cm^2dyn^{-1}x10^{-6}$) and overweight children (18.59±8.08 $cm^2dyn^{-1}x10^{-6}$) ($p<0.001$). AD was significantly higher in obese (0.010±0.005%) children compared to normal weight (0.0036±0.004%) and overweight children (0.009±0.005%) ($p<0.001$). ASβ index and PSEM had inverse trends compared with atrial strain and AD. The results showed that ASβ index had decreasing trend with BMI such that normal weight children had higher value (9.26±6.17), compared to overweight (3.48±2.06) and obese children (2.67±1.15; p<0.001). Same trends occurred for PSEM; the highest value was in normal weight children (7.52±4.76 kPa) and then decreased when BMI increased. SBP increased with BMI significantly (p<0.001) but DBP did not change (p=0.143). Both AOS and AOD significantly increased with BMI (Table I).

Table I also shows the Tukey post-hoc test which indicated the significant differences in different pairs of groups. The results showed that all study parameters except DBP and AOD were different in the normal weight children compared to the overweight and obese group.

Table II shows the linear regression analysis which predicted the effect of age, sex and BMI on aortic stiffness parameters in all participants, in obese or overweight and in normal weight children. In the case of obese or overweight children, analyses revealed that age, sex and BMI predicted 2.6%, 5.0%, 6.0%, 7.0%, 17.6%, 8.3%,41.4% and 34.0% of changes in atrial strain, AD, ASB index, PSEM, SBP, DBP, AOS and AOD, respectively. We also observed that age was the only predictor that had a significant effect on PSEM, DBP and AOS in obese or overweight children.

Table I. Aortic stiffness parameters, blood pressure and aortic diameters according to body mass index groups.

Variables	Groups	Mean ± SD	p	Normal vs. overweight	Normal vs. obese	Overweight vs. obese
AS (%)	Normal	7.06 ± 3.77	<0.001	<0.001	<0.001	0.363
	Overweight	18.59 ± 8.08				
	Obese	20.70 ± 5.04				
AD ($cm^2dyn^{-1}x10^{-6}$)	Normal	0.0036 ± 0.003	<0.001	<0.001	<0.001	0.238
	Overweight	0.009 ± 0.005				
	Obese	0.010 ± 0.005				
ASβ index	Normal	9.26 ± 6.17	<0.001	<0.001	<0.001	0.246
	Overweight	3.48 ± 2.06				
	Obese	2.67 ± 1.15				
PSEM (kPa)	Normal	7.52 ± 4.76	<0.001	<0.001	<0.001	0.238
	Overweight	3.01 ± 1.66				
	Obese	2.34 ± 1.01				
SBP (mm Hg)	Normal	103.49 ± 5.30	0.006	0.005	0.015	0.909
	Overweight	111.71 ± 11.74				
	Obese	113.08 ± 14.55				
DBP (mm Hg)	Normal	63.80 ± 4.70	0.143	0.023	0.435	0.975
	Overweight	66.83 ± 5.66				
	Obese	68.40 ± 11.63				
AOS (mm)	Normal	20.09 ± 3.01	<0.001	<0.001	<0.001	0.222
	Overweight	24.05 ± 3.02				
	Obese	25.10 ± 3.04				
AOD (mm)	Normal	18.78 ± 2.90	0.005	0.026	0.003	0.429
	Overweight	20.36 ± 2.94				
	Obese	20.86 ± 3.00				

AD: aortic distensibility, AOD: aortic diameter in diastole, AOS: aortic diameter in systole, AS: aortic strain, ASβ index: aortic stiffness beta index, DBP: diastolic blood pressure, PSEM: pressure strain elastic modulus, SBP: systolic blood pressure.

Table II. The effect of the age, sex and body mass index on aortic stiffness parameters, blood pressure and aortic diameters.

Parameters	Factors	All participants				Obese or overweight children				Normal body mass index			
		Standardized β	t	P	R^2	Standardized β	t	P	R^2	Standardized β	t	P	R^2
	Age	-0.076	-0.937	0.351		-0.68	-0.402	0.690		-0.193	-1.214	0.231	
AS	Sex	0.031	0.407	0.685	50.7%	0.017	0.100	0.921	2.6%	0.026	0.173	0.864	3.5%
	BMI	0.732	9.515	<0.001		0.163	0.082	0.285		0.126	0.773	0.432	
	Age	-0.183	-1.987	0.05		-0.223	-1.399	0.187		-1.171	-1.072	0.289	
AD	Sex	0.04	0.455	0.65	36.2%	0.013	0.081	0.936	5.0%	0.101	0.680	0.500	3.3%
	BMI	0.636	7.269	<0.001		0.106	0.716	0.478		0.047	0.297	0.768	
	Age	0.175	1.808	0.074		0.128	0.773	0.443		0.321	2.072	0.044	
ASB index	Sex	-0.067	-0.73	0.467	29.6%	0.037	0.229	0.820	6.0%	-0.057	-0.400	0.691	8.9%
	BMI	-0.573	-6.236	<0.001		-0.222	-1.500	0.141		-0.121	-0.781	0.437	
	Age	0.18	1.864	0.065		-0.199	1.209	0.233		0.309	1.990	0.053	
PSEM	Sex	-0.061	-0.67	0.504	30.00%	0.027	0.169	0.866	7.0%	-0.049	-0.341	0.735	8.2%
	BMI	-0.578	-6.306	<0.001		-0.204	-1.387	0.172		-0.141	-0.990	0.365	
	Age	0.228	2.296	0.024		0.316	2.038	0.047		-0.096	-0.614	0.543	
SBP	Sex	-0.012	-0.125	0.901	25.60%	0.045	0.296	0.769	17.6%	-0.200	-1.376	0.176	6%
	BMI	0.384	4.063	<0.001		0.195	1.412	0.165		-0.051	-0.377	0.745	
DBP	Age	0.032	0.297	0.767		0.126	0.769	0.446		-0.183	-1.254	0.216	
	Sex	0.114	1.105	0.272	10.20%	0.128	0.796	0.430	8.3%	0.104	0.771	0.445	19.2%
	BMI	0.271	2.608	0.011		0.170	0.166	0.250		-2.299	-2.299	0.026	
	Age	0.34	3.998	<0.001		0.564	4.318	<0.001		0.189	1.183	0.234	
AOS	Sex	0.048	0.596	0.553	45.4%	0.106	0.823	0.415	41.4%	-0.015	-1.000	0.921	3%
	BMI	0.464	5.734	<0.001		0.100	0.854	0.398		0.057	-0.357	0.713	
	Age	0.407	4.076	<0.001		0.537	0.876	<0.001		0.223	1.442	0.156	
AOD	Sex	0.034	0.36	0.72	24.8%	0.085	0.027	0.534	34%	-0.022	-0.148	0.833	4.4%
	BMI	0.163	1.71	0.091		0.616	0.126	0.900		-0.088	-0.558	0.580	

AD: aortic distensibility, AOD: aortic diameter in diastole, AOS: aortic diameter in systole, AS: aortic strain, ASB index: aortic stiffness beta index, DBP: diastolic blood pressure, PSEM: pressure strain elastic modulus, SBP: systolic blood pressure.

Discussion

The results of the present study showed that atrial strain, AD increased by BMI when ASβ index and PSEM decreased. Among the predicted variables, BMI had a significant effect on atrial strain, AD, ASβ index and PSEM in all participants; and age was the only predicted variable that had a significant effect on PSEM, DBP and AOS in obese or overweight children.

Skilton et al.[21] reported that AS was affected by a few parameters such as; sex, hypertension, smoking, dyslipidemia, chronic inflammatory disease, aging, increased weight, a lower height and several studies have shown changes in AS associated with obesity and have found similar findings with the present study.[4,22] AS is evaluated with numerous parameters, such as atrial strain, AD, ASB index, PSEM, pulse wave velocity (PWV) and augmentation index (AIx).[23] The AIx is a measure of systemic AS derived from the ascending aortic pressure wave form and is inversely associated with body height but is not associated with age, sex or weight.[24] Obesity introduces several structural and hemodynamic changes which cause fat deposition around the vessel wall and changes in compliance; in addition some mediators such as endothelin and nitric oxide, may lead to vasodilatation in resistance vessels.[25] Kulsum-Mecci et al.[4] conducted a study to find the effect of obesity and hypertension on AS using PWV. They found higher PWV in obesity and in hypertension, as well as in combination. They also found a "paradoxical" decrease in PWV with obesity when Acree et al.[26] showed an unexpected result in adults consistently that show higher PWV with obesity.[4] Earlier puberty leading to earlier maturation to peak arterial compliance and increased body size in obese children is one of the hypotheses for the paradoxical decrease.[25]

Hudson et al.[27] showed that obese children were more likely to suffer from a significant damage to AS parameters. They demonstrated that there was a positive correlation between arterial pressure and aortic elasticity independent of obesity. In contrast to our findings that AD was more prevalent in overweight or obese children, changes in AS parameters are a complex phenomenon characterized by a decrease in the distensibility of the large arteries.[24] Both Koopman et al.[28] and Sen et al.[29] reported that obese children had an increase of PWV and changes in AS parameters compared with normal BMI children. In this regard, it could be suggested that obesity can have a significant impact on arterial changes and could play an critical role in the pathophysiology of macrovascular disease.

Dangardt et al.[18] found that obesity had substantial changes in AS parameters as early as 14 to 19 years of age, beyond what was observed in the controls. The degree and duration of obesity are suggested to be important factors for determining the cardiovascular changes. This finding might indicate that obesity initially adapted to accommodate the larger blood volume, generated by the marked increase in fat mass, by overall 'vasodilatation' and increased cardiac functions. But, it seems that there is a limit to this physiological adaptive response; when the limit is reached during adolescence, diastolic blood pressure increases, and it results in the loss of the previously augmented cardiac output effect, evidenced by the observed increase of arterial stiffness.[30] Adiposity itself may have a driving influence on vasculature leading to the development of hypertension.[31] It is also known that conventional risk markers are frequently unstable across adolescence. Hudson et al.[27] found no association between AS parameters and stage of puberty but found that AS parameters change by age. Haraguchi et al.[32] found that AS parameters were significantly related to obesity. It is noteworthy that in addition to hypertension a change in AS also may contribute to the development of cardiac hypertrophy in the obese population.[33] Abdominal adiposity that is measured with a simple clinical tool such as abdominal fat, alone or combined with hypertriglyceridemia, remains a good cardiovascular predictor.[33]

The main limitation of the study was the small sample size due to a single center data collection. But despite this, we believe the study is important as it applied certain measures of AS such as aortic strain, AD, ASβI, and PSEM instead of PWV and AIx.

We conclude that aortic strain and AD increased by BMI increased when ASB index and PSEM decreased. The study also concluded that systolic blood pressure was increased with BMI but diastolic blood pressure did not change. Systolic and diastolic diameters of the aorta increased in obese children. This suggests that AS is a predictor for future heart diseases and children with overweight or obese status must be closely followed to prevent these consequences.

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Ethical approval

The study was approved as a project proposed to the Children and Adolescent Health Research Center by the Ethics Committee of Zahedan University of Medical Sciences, Zahedan, Iran (IR.ZAUMS.REC.1400.095, 2021-5-26).

Author contribution

The authors confirm contribution to the paper as follows: study conception and design: NMN, MNM; data collection: MNM; analysis and interpretation of results: AT; draft manuscript preparation: AT. All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

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