

Table 3. Age-adjusted stepwise multiple linear regression and Pearson correlation analysis

Dependent variables	Independent variables	$\beta \pm SE$	p	r coefficients	p
cIMT					
	Waist Circumference	0.004±0.0001	<0.001	0.728	<0.001
FMD %					
	Waist Circumference	0.07±0.002	<0.001	-0.521	<0.001
	The presence of LBW	-0.028±0.01	<0.04	-0.255	<0.04

cIMT = carotid intima-media thickness, FMD = flow-mediated dilatation, LBW= low birth weight

OP-086**Association between e-NOS gene Polymorphisms and Cardiac Anomaly in Children with Down Syndrome**

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Background: Down syndrome (DS) is a genetic disorder that results in extra genetic material from chromosome 21 and seen in patients with congenital heart defects (CHD). Approximately 40%-50% of patients with Down syndrome have a heart defect. CHD affects 6–8 babies in every 1000 live births and connect to fetal loss. It was reported that the frequencies of CHD were ranging from 16-65% in DS.

The gene which encoding the endothelial isoform of nitric oxide synthase (eNOS) is placed in the long arm of chromosome 7 and includes 26 exons spanning 21 kb of genomic DNA. Nitric oxide (NO) is synthesized from L-arginine by eNOS. To the best of our knowledge there is no study to determine the relation between CHD and eNOS polymorphisms in DS cases. So, we aim to investigate the relationship between eNOS polymorphisms (exon 894 G/T, promoter -786T/C and intron G10T) and cardiac lesions to determine whether this polymorphism was associated with CHD in cases with DS.

Material And Methods: 50 DS cases (22 girls and 28 boys) were included in the current study. The study was confirmed by local ethics committee. In all participants, transthoracic M-mode, two-dimensional, pulsed-wave, continuous-wave and color Doppler echocardiographic examinations were performed using a General Electric Vingmed Vivid 7 (GE Vingmed Ultrasound AS, Horten, Norway) using 2.5-3.5MHz transducers. Blood samples were collected in tubes containing ethylenediamine tetra-acetic acid (EDTA) and total DNA was isolated via phenol-chloroform extraction methods. The eNOS single nucleotide polymorphisms (SNPs) were detected by PCR-RFLP methods.

Results: The mean age of cases was 27.78 ± 31.02 month old. The cardiac evaluation results of cases were: 11 normal, 5 with total atrioventricular septal defect (AVSD), 9 with atrial septal defect (ASD), 9 with ventricular septal defect (VSD), 9 with patent ductus arteriosus (PDA)+ASD, 2 with PDA + Aortic Regurgitation (AR)+ Mitral Regurgitation (MR), 2 with tetralogy of Fallot (TOF) and 3 with ASD+VSD. According to cardiac lesions, the distributions of eNOS genotypes were given in Table 1. The differences between cases who have not a cardiac lesions (named normal in the text) and cases with ASD, PDA+ASD and PDA+AR+MR were meaningful for eNOS promoter (-786T/C) polymorphism (p<0.05). For eNOS exon (894 G/T) polymorphism, the differences among normal and cases with Total ASVD, ASD, VSD, PDA+ASD, PDA+AR+MR and ASD+VSD were meaningful (p<0.05). Conversely these two polymorphisms, there were not a significant differences between normal and cases with at least one cardiac lesion for Intron G10T polymorphism (p>0.05).

Conclusion: It may be said that there is a relation between CHD and both of eNOS promoter -786T/C and exon 894 G/T polymorphisms in cases with DS. eNOS polymorphisms may be used as a tool to obtain preliminary knowledge about the risk of CHDs during prenatal routine screening in DS cases.

Table 1. Distribution of eNOS genotypes according to cardiac lesions.

	Promoter -786T/C			Exon 894 G/T			Intron G10T		
	TT	TC	CC	GG	GT	TT	GC	GT	TT
Cardiac Anomalies									
Normal	6	4	1	9	2	0	9	2	0
Total ASVD	2	3	0	1	3	1	3	2	0
ASD	1	3	5	2	6	1	8	1	0
VSD	3	5	1	1	8	0	7	2	0
PDA+ASD	1	4	4	1	7	1	6	3	0
PDA	0	0	2	0	2	0	1	1	0
TOF	0	2	0	1	1	0	1	1	0
ASD+VSD	0	2	1	0	3	0	2	1	0
Total	13	23	14	15	32	3	37	13	0

ASD; atrial septal defect, ASVD; Atrioventricular septal defect, PDA; Patent ductus arteriosus, TOF; Tetralogy of Fallot, VSD; Ventricular septal defect

Echocardiography**OP-087****Effect of Obesity on Endothelial Function and Subclinical Atherosclerosis in Children**

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Backgrounds: We aimed to measure flow-mediated dilation (FMD), carotid intima-media thickness (cIMT), and to evaluate the effects of waist circumference (WC), and body mass index Z (BMI-Z) score on these parameters in obese children.

Methods: This case-control cross-sectional study included 70 obese and 40 non-obese children aged 7-14 years who presented with various complaints and had no concomitant diseases. FMD and cIMT were measured in all subjects and correlated with anthropometric and biochemical factors.

Results: WC, BMI-Z score, systolic and diastolic blood pressure (BP), triglyceride (TG) and insulin concentrations, and homeostatic model assessment (HOMA) index were significantly higher, whereas high density lipoprotein (HDL) -cholesterol concentration was significantly lower in the obese than in the non-obese group. FMD values were significantly lower, whereas cIMT values were significantly higher in obese than in non-obese subjects. FMD negatively correlated with WC, BMI-Z score, serum insulin level, HOMA, systolic BP, triglyceride but positively with HDL-cholesterol. cIMT positively correlated with WC, BMI-Z score, serum insulin level, HOMA, systolic BP, triglyceride but negatively with HDL-cholesterol.

Conclusions: Increased WC, BMI-Z score, serum insulin level, HOMA, systolic BP, triglyceride and decreased HDL-cholesterol in obese children contribute to endothelial dysfunction and early subclinical atherosclerosis compared to their normal weight peers.