Table 3. Intergroup comparison of atrial conduction times, oxidative stress and inflammation markers

	Patients (n=43)	Controls (n=50)	P value
cPA lateral, ms	69.8±10.4	62.2±8.9	<0.001
cPA septal, ms	50.0±7.5	40.9±6.2	<0.001
cPA tricuspid, ms	45.4±10.2	33.5±5.1	<0.001
Glutathione peroxidase, U/gHb	1.93±1.50	2.04±1.91	0.77
Superoxide dismutase, U/gHb	8677.5±5574.0	9267.7±5538.5	0.61
Catalase, U/gHb	7.8±9.4	8.0±8.7	0.91
Malondialdehyde, nmol/l	17.1±10.3	11.6±7.9	0.005
hsCRP, mg/l	15.7±31.7	4.8±4.7	0.01

cPA, corrected atrial electromechanical delay (time interval from the onset of P wave on surface ECG to the beginning of A wave interval with tissue Doppler imaging); hsCRP, high sensitive C-reactive protein.

Coronary Heart Diseases

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OP-026

Glutathione S Transferases M1 and T1 Genetic Variants are Associated with Coronary Artery Ectasia

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Background: Glutathione S transferases (GSTs) are enzymes responsible for the metabolism of numerous xenobiotics and are known to be polymorphic in humans. Glutathione S-transferases (GSTs) detoxify environmental agents which influence the onset and progression of disease. Carcinogenic and toxic molecules produce DNA adducts that contribute to the development of atherosclerosis. Genetic polymorphisms of xenobiotic-detoxified enzymes, which control the level of DNA adducts, may affect both enzymatic activity and individual susceptibility to coronary artery disease (CAD). Dysfunctional detoxification enzymes are responsible for prolonged exposure to reactive molecules and can contribute to endothelial damage, an underlying factor in CAD. Coronary artery ectasia (CAE) is defined as local or generalized aneurysmal dilatation of the coronary arteries. Although the etiology of CAE has not been identified completely, the most frequent cause is coronary atherosclerosis. Endothelial dysfunction and enzymatic destruction of media layer plays an important role in the pathogenesis of coronary ectasia. We aimed to examine the association between GSTM1, T1 gene polymorphisms and CAE.
Methods: Our study was carried out in a total of 90 cases diagnosed with CAE and a total of 91 population-matched healthy controls in respect to age and genders. After DNA isolation, polymorphisms were analyze using Polymerase Chain Reaction-Restriction Fragment Length Polymorphism (PCR-RFLP) methods. Distribution of the GSTM1 and GSTT1 polymorphism genotypes were determinated as Wild (*1*/1) genotype (Normal genotype) and null (*0*/0) genotype (homozygous deletion). In our study CAE group was also divided into subgroups in respect to herbicide exposure.
Results: No significant differences were obtained in terms of age, genders, risk factors, biochemical parameters and echocardiographic characteristics between study and control groups. The levels of GSTM1 and T1 polymorphisms were statistically higher in CAE cases than controls (p<0,05). Genotype distribution and the levels of GST enzymes of CAE and control groups shown in the table. GSTM1 and T1 homozygous mutations were detected significantly higher in those with exposure to herbicides in patients with coronary artery ectasia group.
Conclusions: To our knowledge this is the first study examining the polymorphisms encoding GST enzymes polymorphisms in cases with CAE. Our results suggest that the deletions in GST genes associated with patients CAE. We think the data obtained in this study can be used to direct people who have deletions in GST genes to avoid chemicals and herbicide exposure.

	KAE		Control		P
	n	%	n	%	
*1/*1 genotype (GSTM1)	47	52,2	65	71,4	0,008
*0/*0 genotype (GSTM1)	43	47,8	26	28,6	
*1/*1 genotype (GSTT1)	67	74,4	81	89	0,011
*0/*0 genotype (GSTT1)	23	25,6	10	11	

	Herbisit (+) KAE (n:43)		Herbisit (-) KAE (47)		P
	n	%	n	%	
*1/*1	8	18,6	39	83	≤0,001
*0/*0	35	81,4	8	17	

	Herbisit (+) KAE (n:43)		Herbisit (-) KAE (n:47)		P
	n	%	n	%	
*1/*1	25	58,1	42	89,4	0,001
*0/*0	18	41,9	5	10,6	

OP-027

The Relationship between Microalbuminuria and Isolated Coronary Artery Ectasia

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Introduction: Coronary artery ectasia (CAE) is localized or diffuse swelling of epicardial coronary arteries 1.5 times the diameter of the adjacent normal coronary segment. Atherosclerosis, congenital causes, inflammatory or connective tissue disorders are among the probable etiologies however exact etiology remains unclear. In some studies, CAE was shown to be a generalized disease affecting other vascular beds. Microalbuminuria is the subclinical increase in urinary albumin which is 30-300 mg/day in a spot urine check and is related to increased morbidity and mortality in cardiovascular disease. The aim of our study is to find out the relationship between levels of urinary microalbumin and isolated coronary artery ectasia.
Methods: 58 patients with isolated CAE and 57 control subjects with normal coronary arteries (NCA) were included in the study. Previous history of myocardial infarction and percutaneous intervention, left ventricular hypertrophy, left ventricular dysfunction (EF <50%), moderate-severe valvular disease, rhythms other than sinus, congenital heart disease, chronic obstructive lung disease and/or cor pulmonale, chronic systemic illness, active infection, renal failure, neoplastic disease, antioxidant drug usage and alcohol abuse were the exclusion criteria. Midstream random urine samples were collected in the morning and microalbuminuria was determined by immunoturbidimetric method. The results were given as albumin / creatinine ratio (mg/mg). The values between 0.03 and 0.3 were defined as microalbuminuria.
Results: The mean age was 60,55±9,77 in CAE and 57,32±8,30 in NCA groups,respectively. There was not any statistically significant difference between groups according to the cardiovascular risk factors like gender, hypertension, smoking, hyperlipidemia, diabetes mellitus and family history of CAD and the drugs used. Urinary albumin to creatinine ratio was 0,036±0,040 in isolated CAE and 0,018±0,013 in the control group, respectively, and the difference was statistically significant (p=0.002).
Conclusions: Microalbuminuria is a well established risk factor for cardiovascular morbidity and mortality. In our study we showed that urinary microalbumin levels