

hypertension, diabetes, history of heart disease and smokers were excluded. A control group of 81 patients between the ages of 20-50 were chosen from various hospitals in Ankara. All patients underwent transthoracic echocardiography. Left ventricular septal, lateral, anterior and inferior wall velocities were recorded with tissue Doppler. The patients were divided into two groups: Affected by lead in Group 1 and control in Group 2.

Results: There was no significant difference between demographic characteristics of the patients as well as between left ventricular ejection fraction. (Table 1) Left ventricular lateral, septal, anterior and inferior wall velocities were significantly decreased in group 1. (Table 1) Blood lead levels were measured between 3.98 to 75 mcg/dL in group 1. Decrease in velocity, was found to be more pronounced especially in those blood lead level was 50 mcg/dL or more

Conclusions: Pb induced cardiotoxicity has never been investigated yet and the exact mechanism of Pb associated cardiotoxicity has not been studied. In a recently designed study, the potential effect of Pb to induce cardiotoxicity in vivo and in vitro rat model was investigated. The molecular mechanisms and the role of aryl hydrocarbon receptor (AHR) and its related gene and cytochrome P4501A1 (CYP1A1) in Pb(2+)-mediated cardiotoxicity was explored. In this study, we have demonstrated the need for a detailed examination with echocardiography in patients having lead exposure. Echocardiography may be an important diagnostic tool for the detection of myocardial damage associated with lead toxicity.

Table 1. Demographic data in Group 1 and Group 2 and Echocardiographic measurements in Group 1 and 2

	Group 1 (n=106)	Group 2 (n=81)	p
Age (year)	38 ± 12.2	26.1 ± 13.1	0.049
LA (cm ²)	4.61 ± 0.71	4.72 ± 0.53	0.494
LVED (cm)	4.82 ± 0.56	4.73 ± 0.44	0.521
LVES (cm)	3.20 ± 0.48	3.02 ± 0.52	0.176
EF (%)	61.82 ± 6.93	66.83 ± 7.20	0.12
Lateral systolic velocity(m/s)	10.53 ± 1.32	15.64 ± 1.23	< 0.001
Anterior systolic velocity(m/s)	8.1 ± 1.14	13.0 ± 0.13	< 0.001
Inferior systolic velocity(m/s)	9.2 ± 0.18	14.1 ± 0.16	< 0.001
Septum systolic velocity (m/s)	9.11 ± 3.79	15.93 ± 2.96	< 0.001

Group 1: Patients affected by lead, Group 2: control group LVED: Left ventricle end diastolic, LVES: Left ventricle end systolic, EF: Ejection fraction, RA: Right atrium

PP-226

Assessment of Subclinical Left and Right Ventricular Dysfunction by Using Isovolumic Acceleration in Patients with Dipper and Non-dipper Hypertensives

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Background: Patients with non-dipper hypertension have increased cardiovascular mortality and morbidity compared with dipper hypertensives. Myocardial acceleration during isovolumic contraction (IVA) has been used as a novel tissue doppler parameter for assessment of left and right ventricular systolic functions regardless of preload and afterload dependency.

Objectives: We aimed to compare IVA and tissue doppler echocardiographic parameters of dipper and non-dipper hypertensive patients to predict subclinical right and left ventricular systolic dysfunction.

Methods: 45 normotensive healthy volunteers (20 men, mean age 43 ± 9 years), 45 dipper hypertensive (27 men, mean age 45 ± 9 years) and 45 non-dipper hypertensive patients (25 men, 47 ± 7 years) were enrolled in the study following 24 hour ambulatory blood pressure monitoring. All the participants underwent conventional and tissue Doppler echocardiographic (TDI) examination. Myocardial velocities of left ventricular (LV) septal, lateral mitral annulus and tricuspid lateral annulus were recorded. IVA was measured by dividing the peak myocardial isovolumic contraction velocity (IVV) by isovolumic acceleration time (AT).

Results: Non-dipper hypertensives indicated lower LV IVA (2.2 ± 0.4 m/sec² versus 2.8 ± 1.0 m/sec², p < 0.01) and RV IVA values (2.8 ± 0.8 m/sec² versus 3.5 ± 1.0 m/sec², p = 0.012) compared with dippers although the other conventional and TDI echo parameters were similar. On the other hand, normotensives had increased LV IVA (p < 0.001) and RV IVA (p < 0.001) values compared with dipper and non-dipper hypertensives. LV mass index (p = 0.001), LV interventricular septal thickness (p = 0.002) and LV MPI (p < 0.001) were negatively correlated with LV IVA. LV interventricular septal thickness (p = 0.002), LV mass index (p = 0.001) and RV MPI (p < 0.001) were significantly negative correlated with RV IVA.

Conclusion: The present study revealed that non-dipper hypertensives have reduced left and right ventricular IVA values indicating subclinical systolic impairment compared with dipper hypertensives.

PP-227

The Relationship Between Neutrophil / Lymphocyte Ratio and Myocardial Ischemia Detected in Dobutamine Stress Echocardiography

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Aim: Neutrophil / lymphocytes (N / L) ratio is an indicator of systemic inflammation. It is shown that N/L ratio is a predictor of cardiac events and mortality at acute coronary syndrome and stable coronary artery disease. The relationship between presence and extent of myocardial ischemia with the N / L ratio is unknown. In this study, it was investigated whether there is a relationship between myocardial ischemia detected at dobutamine stress echocardiography (DSE) and N / L ratio.

Method: A total of 917 patients who had known or suspected coronary artery disease, had ejection fraction ≥ 40 and were decided to perform DSE for investigating ischemia included to this prospective study between October 2009 and March 2013. Patients were divided into two groups based on their DSE results as positive DSE and negative DSE. Based on the number of ischemic segments detected at DSE, patients were divided into three groups: no ischemia at DSE (DSE negative group), ischemia at 1-3 segments, ischemia at 4 or more segments.

Results: 640 (69,8%) of 917 patients who were included in the study had no ischemia in DSE (DSE negative group, mean age 58,9 ± 9,9 years, 51,6% female). Ischemia was detected at 277 patients (DSE positive group, mean age 60,5 ± 9,6 years, 38,6% female). Demographic and laboratory characteristics of the positive and negative DSE groups are presented in table 1. N/L ratio of DSE positive group was significantly higher than DSE negative group [N/L ratios: 2,6 ± 1,6, 2,2 ± 1,0 (103/mm³) respectively, p < 0,001]. N/L ratios based on the number of ischemic segment are presented at table 2. N/L ratios of patient who had ischemia at 1-3 and 4 and more segments had significantly higher N/L ratios compared to DSE negative group.

Conclusion: A relationship was found between N/L and myocardial ischemia detected at DSE in our study. This finding suggests that underlying ischemia may be responsible from the relationship between N/L ratio and increased cardiovascular mortality.

Table 1. Demographics and laboratory findings of the DSE negative and positive patients.

	DSE negative (n=640)	DSE positive (n=277)	p
Age (year)	58.9 ± 9.9	60.5 ± 9.6	0.03
Female gender n (%)	330 (51.6)	107 (38.6)	< 0.001
Hypertension n (%)	155 (24.2)	102 (36.8)	0.01
Diabetes mellitus n (%)	176 (27.5)	87 (31.4)	0.26
Smoking n (%)	158 (24.7)	78 (28.1)	0.19
Known coronary artery disease n (%)	148 (23.1)	115 (41.5)	< 0.01
Creatinine clearance (ml/min)	94.4 ± 23.1	94.6 ± 22.1	0.92
Hemoglobin (gr/dl)	14.0 ± 1.1	14.1 ± 1.0	0.44
White blood cell count (10 ³ /mm ³)	7.2 ± 1.6	7.6 ± 1.7	< 0.001
Neutrophil (10 ³ /mm ³)	4.2 ± 1.3	4.7 ± 1.5	< 0.001
Lymphocyte (10 ³ /mm ³)	2.2 ± 0.7	2.1 ± 0.7	0.28
Neutrophil/Lymphocyte ratio	2.2 ± 1.0	2.6 ± 1.6	< 0.001

DSE: Dobutamine stress echocardiography

Table 2. Comparison of some hematological parameters according to number ischemic segments

	DSE negative (n=640)	1-3 segment ischemia (n=180)	4 and above segment ischemia (n=97)	p
Neutrophil (10 ³ /mm ³)	4.2 ± 1.3	4.6 ± 1.5*	4.8 ± 1.5*	< 0.001
Lymphocyte (10 ³ /mm ³)	2.2 ± 0.7	2.1 ± 0.8	2.1 ± 0.7	0.56
Neutrophil/Lymphocyte ratio	2.2 ± 1.0	2.6 ± 1.5*	2.7 ± 1.7*	< 0.001

* Comparison of DSE negative group according to Post hoc Tukey test p < 0.05 DSE: Dobutamine stress echocardiography