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Original Article

Evaluation of myocardial performance index in smokers

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Abstract

Aim: The aim of this study was to evaluate MPI and left ventricular (LV) functions in patients without cardiac risk factors except smoking. Smoking is associated with increased cardiovascular events. Myocardial performance index (MPI) is a noninvasive tissue doppler technique that provides information about left ventricular systolic and diastolic functions.

Material and Methods: A total of 50 smokers and 50 healthy volunteers were consequently included in the study. All participants underwent echocardiographic examinations, including MPI measurements.

Results: We found that MPI values were significantly higher in the smoker group (0.52 ± 0.08, 0.42 ± 0.07, p< 0.001). E wave (0.73 ± 0.18, 0.84 ± 0.25, p = 0.018), IVRT (77.52 ± 11.90, 61.80 ± 12.85, p <0.001), IVCT (60.84 ± 15.67, 53.16 ± 10.68, p = 0.005) values were significantly lower in the control group.There was a positive correlation between packet-year and MPI (r = 0.525, p<0.001)

Conclusion: In our study, we observed increased MPI in smokers, which was shown to be associated with left ventricular function. We think that MPI could be used as an easy method to determine subclinical left ventricular dysfunction in healthy smokers.

Keywords: Myocardial performance index; smoker; tissue doppler echocardiography

INTRODUCTION

Cigarette consumption has become a major health problem worldwide. Smoking is an independent risk factor for coronary artery disease. In a study has shown a strong relationship between smoking and cardiovascular diseases (1). Smoking has also been shown to cause myocardial dysfunction with different mechanisms such as activation of the autonomic nervous system, inflammation, endothelial system dysfunction and oxidative stress (2-5).

The myocardial performance index (MPI), which can be used to analyze both systolic and diastolic cardiac performance, is obtained using tissue Doppler imaging (TDI) (6). Increased MPI has been shown to be prognostic index and independent predictor of cardiac death in various heart diseases and may also provide information about subclinical cardiac dysfunction (7, 8). Based on this information, we aimed to evaluate left ventricular (LV) systolic and diastolic functions by using tissue Doppler echocardiography in patients who smoke but do not have any health problems.

MATERIAL and METHODS

Fifty individuals aged between 18-40 years who smoked and did not have any cardiovascular risk factors other than smoking and fifty healthy non-smoking volunteers were included in the study. Passive non-smokers; Individuals who were not in closed smoking areas (home, workplace, restaurant, cafe) were included in the healthy control group. Physical examination of all individuals was completely normal and none of them had received any treatment before. A pack of cigarette was considered 20 cigarettes. The number of cigarette packets smoked per day was multiplied by the number of years smoked. The number of packets-years was calculated. Patients with the following conditions were excluded from the study: Hypertension, coronary artery disease, diabetes mellitus, objective ischemia findings of coronary artery disease (wall motion impairment in echocardiography, evidence of ischemia in exercise test or myocardial single photon emission computed tomography), heart valve disease, systolic heart failure, rheumatic and collagen tissue diseases, chronic kidney or liver disease, dysrhythmia

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and malignancy. Baseline demographic characteristics, systolic and diastolic blood pressure, heart rate (HR) and smoking duration of all subjects were recorded. Standard resting electrocardiography (ECG) of all individuals was taken in the study. Conventional echocardiography and tissue doppler measurements were performed in all subjects. Subjects included in the study were required not to smoke in the last 8 hours. Informed consent forms were obtained from all participants. Local ethics committee approval was obtained for study. Venous blood samples were obtained from the left antecubital vein after 8 hours of fasting for hemogram and biochemical blood analysis.

Echocardiography

All patients underwent echocardiographic evaluation using Philips IE33 (Philips, Bothell, USA) with a 3.5-MHz transducer in the left side position. Patients were monitored during transthoracic echocardiographic evaluation. Two dimensional, pulsed doppler, continuous wave doppler, color and tissue doppler evaluations were performed using standard echocardiographic techniques. Left atrium (LA) size, LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), Inventricular septum thickness (IVST), posterior wall thickness (PWT) were measured by Mmode method. During apical four-chamber imaging, transmitral flow waves (E and A) were measured by pulse wave Doppler (PWD) method and E / A ratio was calculated. Tissue Doppler imaging was performed with the sample volume placed in the medial and lateral mitral annulus in 4 apical chamber windows. Isovolumetric relaxation time (IVRT), Isovolumetric contraction time (IVCT), Ejection time (ET), early diastolic mitral annular velocity (Em), Late diastolic mitral annular velocity (Am) and peak systolic mitral annular velocity (S) were recorded. IVRT was obtained by measuring the time in milliseconds between the midpoint of aortic closure and the appearance of the E wave resulting from the opening of the mitral valve. IVCT was calculated by measuring the time in milliseconds between the tip of the mitral A wave and the opening of the aortic valve. ET was measured in milliseconds by measuring the duration of the start and end points of the S wave showing the ejection period. The formula used to determine the MPI value is as follows (IVRT + IVCT) / ET (9). Left ventricular ejection fraction (LVEF) was calculated by Simpson and M mode methods and the mean value was recorded.

Statistical analysis

SPSS (SPSS 22.0 software) was used for all statistical analysis. Mean ± standard deviation (SD) for continuous variables and categorical variables were expressed as percentages. In the comparison of categorical variables, Pearson Chi-Square Analysis and Fisher's Exact test, and the relationships between continuous variables were examined by Pearson and Spearman correlation tests. The correlation between packet-year and MPI was tested by Pearson correlation analysis. P <0.005 was considered statistically significant.

RESULTS

Demographic and laboratory parameters of the patients are shown in Table 1.

Table 1. Comparison of clinical and laboratuar parameters between two groups

	Smoker group N:50	Control group N: 50	P Value
Age (year)	33.9 ± 5.78	32.58 ± 5.92	0.241
Sex (Male%)	25(50)	21(42)	0.422
BMI (kg/m²)	26.23 ± 3.99	25.44 ± 4.91	0.380
SBP (mmHg)	122.24 ± 8.32	118.8 ± 8.83	**0.048**
DBP (mmHg)	76.86 ± 7.02	75.92 ± 7.63	0.523
LDL (mg/dL)	120.18 ± 34.02	110.48 ± 25.67	0.144
HDL (mg/dL)	46.98 ± 9.67	50.03 ± 9.85	0.145
TG (mg/dL)	135.83 ± 46.83	133.82 ± 35.58	0.815
Hgb (g/dL)	14.02 ± 1.84	13.68 ± 1.20	0.287
Fasting glucose (mg/dL)	91.93 ± 28.71	88.93 ± 9.87	0.491
Packet-Year	18.1±10.5	-	-
Heart Rate (beats / minute)	81.28 ± 12.32	75.96 ± 9.53	**0.018**

BMI : Body Mass Index SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, LDL : Low-density lipoprotein. HDL : High-density lipoprotein; TG :Triglyceride, Hgb : Hemoglobin

There was no statistically significant difference between the groups in terms of age, sex, body mass index, diastolic blood pressure, LDL, HDL, TG, Hgb, and fasting blood glucose. The systolic blood pressure and HR values were higher in the smoker group compared to the control group and were statistically significant (122.24 ± 8.32,118.8 ± 8.83, p = 0.048; 81.28 ± 12.32, 75.96 ± 9.53, p = 0.018, respectively). Table 2 shows the comparison of the echocardiographic parameters of the groups. There was no significant difference between LVEF, LA diameter, LV end-systolic and end-diastolic diameters, A wave, E / A, DT and ET, but E wave (0.73± 0.18, 0.84 ± 0.25, p = 0.018) was lower in the smoker group, IVRT (77.52±11.90, 61.80 ±12.85, p <0.001), IVCT (60.84±15.67, 53.16±10.68, p = 0.005) and MPI (0.52±0.08, 0.42±0.07, 0.42±0.07, p <0.001) values were significantly lower in the control group. As shown in Figure 1, there was a positive correlation between MPI and pack-year (r = 0.525, p <0.001).

Table 2. Comparison of clinical and laboratuar parameters between two groups

	Smoker group N:50	Control group N: 50	P Value
Ejection Fraction (%)	61.84 ± 2.41	61.44 ± 2.85	0.451
Left Atrial diameter (mm)	34.52 ± 5.25	34.08 ± 2.93	0.603
LVEDD (mm)	46.72 ± 2.73	46.44 ± 3.78	0.672
LVESD (mm)	23.80 ± 4.58	24.66 ± 3.83	0.316
E wave (cm/sec)	0.73 ± 0.18	0.84 ± 0.25	**0.018**
A wave (cm/sec)	0.66 ± 0.11	0.64 ± 0.12	0.425
E/A	1.15 ± 0.36	1.29 ± 0.42	0.078
DT (ms)	143.7 ± 19.33	146.02 ± 23.94	0.595
IVRT (ms)	77.52 ± 11.90	61.80 ± 12.85	**<0.001**
IVCT (ms)	60.84 ± 15.67	53.16 ± 10.68	**0.005**
ET (ms)	262.96 ± 23.0	267.68 ± 23.32	0.311
MPI	0.52 ± 0.08	0.42 ± 0.07	**<0.001**

LVEDD : Left ventricular end-diastolic diameter (cm), LVESD : Left ventricular end-systolic diameter (cm), E : Mitral early diastolic velocity, A : Late mitral diastolic velocity, DT : Deceleration time, IVRT : Isovolumetric relaxation time, IVCT : Isovolumetric contraction time, ET : Ejection Time, MPI : Myocardial performance index

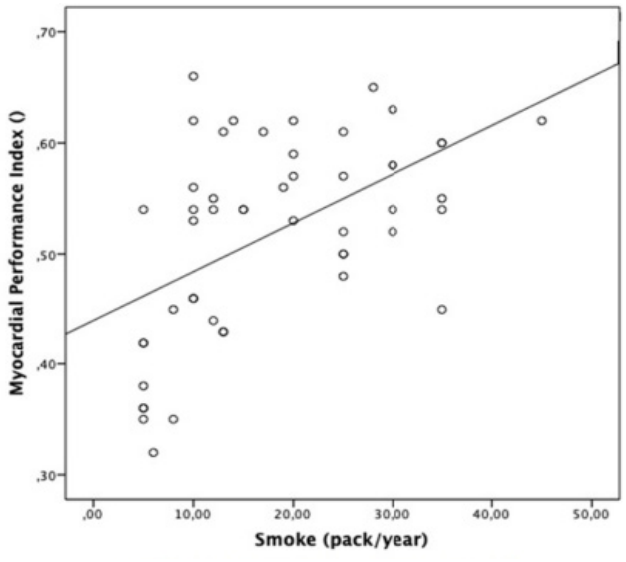

Figure 1. Correlati on between MPI and pack-year

DISCUSSION

In our study, it was shown that MPI, which shows left ventricular myocardial systolic and diastolic functions and is associated with prognosis, is higher in smokers than healthy controls. At the same time, this effect was found to be related to smoking intensity. Smoking is one of the most important modifiable risk factors for cardiovascular diseases (10). The diastolic stage of the cardiac cycle is an active process. It is usually the first affected stage when developing cardiac dysfunction (11). It is known that coronary flow dynamics is impaired in smokers.

As a result of this change in the coronary circulation, the oxygen supply and demand balance of the myocardium is disrupted and causes diastolic dysfunction. In a study by Jia-Ping Wu et aL, It was found that smoking exposure causes myocardial fibrosis in mice (12,13). There are many pathophysiological mechanisms of smoking that can be explained as causing myocardial dysfunction. If we look at these mechanisms; I) decreases oxygen carrying capacity by increasing carbon monoxide in the blood; II) triggers endothelial dysfunction, III) increase in sympathetic activation due to the release of catecolamine, IV) development of coronary spasm may be among the reasons (3,4). It has also shown that smoking has detrimental effects on the mechanism of DNA damage and cardiomyocyte cell function. Similarly, in vitro studies and some animal models have shown mitochondrial damage to the myocardium by smoking (14). MPI was associated with both systolic and diastolic myocardial function assessed using conventional and newer echocardiographic measurements (13). MPI has prognostic value in some heart diseases. MPI has been shown to be associated with subclinical systolic and diastolic dysfunction in various disease groups (15,16). In addition, it has been shown that MPI can provide information correlated with LVEF systolic measurements and global longitudinal strain using strain echocardiography (17). In previous studies, subclinical myocardial dysfunction in healthy smokers was demonstrated by some electrocardiographic and echocardiographic parameters (18). In our study, it was observed that MPI value was higher in smokers. In addition, there was a strong positive correlation between smoking intensity and MPI. The main advantages of this method are that they are easy to apply, can be performed in every eco-laboratory, do not require special equipment and programs and are not time consuming.

In some animal studies, smoking has been shown to cause increased edema and decreased respiration and phosphorisation, and DNA damage in myocardial mitochondria, and is called "smoke cardiomyopathy" (14). This condition causes an increase in left ventricular end-diastolic pressure. IVRT prolongation may result from prolongation of diastolic filling time, increased LV end diastolic pressure, or defect in diastolic relaxation (19). In our study, IVRT was found to be longer in smokers compared to the control group, which may be an early indicator of LV diastolic dysfunction in smokers.

LIMITATION

There are many limitations of our study. First; the number of individuals included in the study was inadequate and the study was performed in a single center, Second; The MPI parameter measured by this tissue doppler is not correlated with a more advanced imaging method such as strain echocardiography, Third; we did not perform blood CO level measurements of study population with known effects on cardiac functions in our study. Finally, echocardiographic evaluation is performed by a single cardiologist.

CONCLUSION

In this study, it was concluded that smoking may adversely affect LV diastolic and systolic functions without other cardiovascular risk factors. Non-invasive methods such as Doppler echocardiography and MPI, which can be performed in smokers in the early period, can be followed up for individuals at risk for subclinical cardiac dysfunction. Smokers can be encouraged to change their lifestyles by using these easily applicable and simple methods.

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