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Echocardiography

The Effect of Smoking on Myocardial Performance Index in Middle-Aged Males after First Acute Myocardial Infarction

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Background: Cigarette smoking is associated with increased rates of coronary artery disease and acute myocardial infarction (MI). Paradoxically, smokers had lower mortality after MI. The purpose of this study was to evaluate the effect of chronic smoking on myocardial performance index (MPI) in middle-aged men after an acute MI. Material and methods: A total of 429 patients (325 smokers vs. 104 nonsmokers) presenting with acute ST elevation MI were enrolled in this study. Thrombolysis in myocardial infarction (TIMI) flow of the infarct related artery was measured before and after the primary percutaneous coronary intervention (PCI), and Gensini score was also calculated. Conventional echocardiography and tissue Doppler echocardiography (TDI) were performed within 48–72 hours after onset of chest pain. Peak early (Em) and late (Am) diastolic velocities, peak systolic (Sm) mitral annular velocities and time intervals were recorded with TDI. The MPI, ratio of Em/Am, and E/Em were calculated. Results: Baseline demographic and angiographic characteristics such as Gensini score, pre and, post PCI TIMI flow were similar in 2 groups. In contrast, LV MPI was preserved among smokers (0.59 ± 0.15 vs. 0.66 ± 0.14, $P = 0.01$), and Em/Am values were also higher in smokers (0.84 ± 0.28 vs. 0.75 ± 0.31, $P = 0.01$). Independent predictors of impaired MPI (≥ 0.60) were determined as nonsmoking status (odds ratio 2.940, 95% CI 0.98–5.83, $P = 0.05$), left anterior descending artery stenosis (odds ratio 3.196, 95% CI 1.73–5.91 $P = 0.001$), and, age (odds ratio 1.12, 95% CI 1.03–1.22, $P = 0.01$). Conclusions: Despite similar demographic and angiographic characteristics, smoker males had a paradoxically better MPI after acute MI. (Echocardiography 2013;30:155-163)

Key words: smoking, Tei index, acute myocardial infarction, tissue Doppler echocardiography

Cigarette smoking is linked to an increased risk of acute myocardial infarction (MI), coronary artery disease (CAD), and death.[1–4] Smokers suffer from MI about 1 decade earlier than nonsmokers.[5] Despite the negative effects of smoking, smokers have a lower death rate, reinfarction, and major adverse cardiac events than nonsmokers after acute MI, especially in patients treated with fibrinolytic therapy.[6,7] This remains known as "the smoker's paradox."[8] Some explanations are younger age, fewer comorbidities, such as diabetes mellitus and hypertension, lower atherosclerotic burden, higher procedural success, and frequent inferior/infero-posterior MI in smokers; however, this paradox is not entirely understood.[6–11]

Myocardial performance index (MPI) is a reliable, noninvasive, feasible, and reproducible parameter. It reflects the combination of systolic and diastolic performance, and it is less influenced by loading conditions, blood pressure, heart rate, and ventricular geometry than conventional echocardiographic parameters, especially ejection fraction (EF).[12–14] The MPI is a powerful independent prognostic factor in the early phase of MI.[15] It is an independent predictor of cardiac events such as cardiac death, cardiogenic shock, arrhythmias, and development of heart failure after MI.[14–16] Higher MPI values (>0.60) represent LV dysfunction in the early phase of MI and help clinicians identify high risk patients.[15] Despite cumulative data about the value of MPI, the association between smoking

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status and MPI in acute MI has not been studied yet.

For this reason, we planned this study to examine the effects of chronic smoking on LV MPI after acute MI in successfully revascularized patients with primary percutaneous coronary intervention (PCI), particularly its association with the smoker's paradox.

Materials and Methods:

Participants:

A total of 429 middle-aged (40–65 years old) male patients presenting with acute ST elevation MI were enrolled between September 2006 and January 2012 from 2 centers (Table I). The smokers (n = 325, Group 1) were compared with 104 nonsmokers (Group 2). All smoker patients included in the study had been smoking more than 10 cigarettes per day. All patients provided written informed consent for participation in the study. The study protocol was approved by the local ethics committee.

Exclusion criteria were the following: ex-smoker patients, diabetes mellitus, female gender, atrial fibrillation, cardiogenic shock, significant cardiac valvular abnormalities, chronic obstructive pulmonary disease, renal or hepatic failure, and inadequate echocardiographic image quality. In addition, patients with no-reflow (being TIMI 0 –I flow after PCI) phenomenon and patients receiving fibrinolytic therapy and needing emergency coronary artery bypass graft operation (CABGO) due to unsuitable coronary anatomy for the PCI were excluded. Finally, we excluded those patients admitted to the hospital after 12 hours of initial symptoms. Furthermore, none of the patients had a history of CAD, percutaneous transluminal coronary angioplasty, and CABGO.

Definitions and Data Collection:

Acute MI is typical chest pain lasting for more than 30 minutes with ST segment elevation >1 mm in 2 or more consecutive precordial or inferior leads.[17] Detailed in-hospital data were prospectively collected including age, risk factors for CAD, peak serial myocardial band fraction of creatine kinase (CK-MB) and peak troponin levels, WBC count, creatinine, and angiographic findings.

Coronary Angiography and PCI:

As our hospital is 7/24 PCI-capable center, primary PCI was preferred as the perfusion strategy for the patients admitted to study hospitals. All angiography and PCI were performed by means of angiography unit (Integris Allura 9; Philips, Eindhoven, The Netherlands). Coronary angiography was performed by the Judkins technique through femoral artery access. Thrombolysis in myocardial infarction (TIMI) flow of the infarct related artery (IRA) was measured before and after the procedure.[18] The severity of CAD was assessed by using the Gensini score.[19] Evaluation of all coronary angiograms was performed by 2 observers, who were blinded to the smoking status. Pre PCI antiaggregant treatment of patients were as follows: oral administration of 300 mg of acetyl salicylic acid and 600 mg loading dose of clopidogrel, an intravenous 70 U/kg bolus dose of unfractionated heparin followed by 12–15 U/kg adjusted according to activated partial thromboplastin time (60–80 sec). Alternatively, enoxoparine was used in most of the patients as an anticoagulant with a dose of 1 mg/kg bid subcutaneously. Tirofiban administration was at the discretion of the physician performing the procedure; however, it was encouraged in case of high thrombus burden. Tirofiban was infused in 2 stages: 0.4 mcg/kg per minute during the first 30 minutes then 0.1 mcg/kg per minute over 24 hours. Other medications are listed in Table I.

Echocardiographic Evaluation:

All patients were evaluated by two-dimensional (2D), pulsed wave Doppler and pulsed-wave tissue Doppler echocardiography (TDI). Echocardiographic examination was performed within 48–72 hours after onset of chest pain by 2 experienced echocardiographers. Patients were examined in the supine and left lateral decubitus position by a Philips Envisor C echocardiograph (Philips Medical Systems, Andover, MA, USA) using a 3.5 MHz transducer. ECG tracing was obtained during echocardiographic examination. The heart was imaged through multiple acoustic windows (parasternal, longitudinal and cross-sectional, apical 4-chamber, and 2-chamber views).

In the apical four-chamber view, the Doppler beam was aligned as perpendicular as possible to the plane of the mitral annulus; the sample volume was placed between the tips of the mitral leaflets during diastole and the following transmitral Doppler parameters were analyzed: peak early (E) and late (A) transmitral filling velocities, the ratio of early to late peak velocities (E/A), and deceleration time of mitral E-wave (EDT).[20] Systolic function was evaluated by measuring wall-motion score index using a 16-segment model with a score from 1 (normal) to 4 (dyskinetic).[21] EF was determined from apical two-chamber and four-chamber views using the Simpson's biplane formula.[22]

The TDI records were obtained on the same echocardiography machine by activating

TABLE I

Demographic Characteristics and Laboratory Findings of the Study Population

Variables	Nonsmoker (n = 104)	Smoker (n = 325)	P
Patient characteristics			
Age	54.6 ± 6.7	53.5 ± 6.8	NS
HT n (%)	16 (15.3)	51 (15.7)	NS
BMI (kg/m^2)	27.1 ± 3.2	26.6 ± 3.5	NS
Family history	95 (29.2)	31 (29.8)	NS
Smoking (pack/year)	–	33.2 ± 13.5	
Hemodynamic parameters			
Systolic BP (mmHg)	107.5 ± 14.4	109.2 ± 15.0	NS
Diastolic BP (mmHg)	68.2 ± 7.3	67.9 ± 9.0	NS
Heart rate (bpm)	75.2 ± 14.0	73.8 ± 11.9	NS
Killip class	1.6 ± 0.6	1.5 ± 0.5	NS
Acute MI characteristics			
MI Location			
Anterior (±lateral) n (%)	53 (51.0)	153 (47.1)	NS
Inferior (±posterior) n (%)	47 (45.2)	158 (48.6)	NS
Other n (%)	4 (3.8)	14 (4.3)	NS
Time intervals			
Time from symptom onset to PCI (min)	258 ± 119	247 ± 133	NS
Time from symptom onset to echo examination (h)	60.2 ± 5.5	61.7 ± 4.8	NS
Angiographic characteristics			
Infarct related artery			
LAD n (%)	55 (52.9)	164 (50.5)	NS
Cx n (%)	30 (28.8)	93 (28.6)	NS
RCA n (%)	19 (18.3)	68 (20.9)	NS
Pre PCI TIMI flow	1.1 ± 0.5	1.1 ± 0.6	NS
Post PCI TIMI flow	2.5 ± 0.5	2.7 ± 0.6	NS
Multivessel disease n (%)	41 (39.4)	118 (36.3)	NS
Gensini score	54.2 ± 39.2	48.3 ± 41.3	NS
Stent usage n (%)	27 (58.7)	72 (56.7)	NS
Complete ST segment resolution after PCI n (%)	72 (69.2)	219 (67.4)	NS
Laboratory characteristics			
Creatinine (mg/dL)	1.07 ± 0.26	1.04 ± 0.40	NS
Peak CK-MB (IU/L)	190.9 ± 160.8	222.1 ± 178.8	NS
Peak troponin (ng/mL)	71.9 ± 39.3	76.2 ± 45.5	NS
WBC on admission ($\times 10^3$/mL)	7.38 ± 5.71	7.98 ± 6.65	NS
Admission medication			
Aspirin, n (%)	12 (11.5)	35 (10.8)	NS
Beta-blocker, n (%)	7 (6.7)	22 (6.8)	NS
ACE inhibitor/ARB, n (%)	10 (9.6)	38 (11.7)	NS
Statin, n (%)	18 (17.3)	50 (15.4)	NS
In-hospital medication			
Aspirin, n (%)	104 (100)	325 (100)	NS
Beta-blocker, n (%)	80 (76.9)	280 (86.1)	NS
ACE inhibitor/ARB, n (%)	88 (84.6)	278 (85.5)	NS
Statin, n (%)	104 (100)	325 (100)	NS
Heparin, n (%)	6 (5.8)	30 (9.2)	NS
LMWH, n (%)	98 (94.2)	295 (90.8)	NS
Tirofiban, n (%)	7 (6.7)	25 (7.7)	NS
Clopidogrel, n (%)	104 (100)	325 (100)	NS

HT = hypertension; BMI = body mass index; BP = blood pressure; MI = myocardial infarction; PCI = percutaneous coronary intervention; LAD = left anterior descending artery; Cx = circumfrex coronary artery; RCA = right coronary artery; TIMI = thrombolysis in myocardial infarction; CK-MB = creatine kinase myocardial band; WBC = white blood cell; ACE = angiotensin converting enzyme; ARB = angiotensin II reseptor blocker; LMWH = low molecular weight heparin. ST resolution = reduction of at least 50% in the Σ ST segment elevation between the pre and post PCI ECGs.

the TDI function and using a 2.5 MHz transducer with a mean frame rate of 105 frame/sec (80–140 frame/sec) in the left lateral decubitus position during shallow respiration or end-expiratory apnea. Guided by the 2D four-chamber view, a sample volume (2 mm) was placed at the septal and lateral corner of the mitral annulus from the apical four-chamber view. The TDI cursor was placed at the anterior and inferior sides of the mitral annulus in the same manner. Care was taken to obtain an ultrasound beam parallel to the direction of the mitral annular motion. Filters were set to exclude high frequency signals, and gains were set to obtain clear tissue signals with minimal background noise. Peak early (Em) and late (Am) diastolic velocities and peak systolic (Sm) mitral annular velocities were recorded. The ratio of Em to Am (Em/Am) and E to Em were calculated.[23] Isovolumetric contraction time (ICT), ejection time (ET), and isovolumetric relaxation time (IRT) were recorded. The MPI was calculated as (ICT + IRT)/ET based on TDI measurements. Mean MPI was calculated as the average MPI of 4 sample sites: anterior, inferior, lateral, and interventricular septum. All measurements were averaged over 3 consecutive cardiac cycles. To assess the intraobserver variability, echocardiographic measurements were repeated in 30 subjects the next day by the same investigator. Echocardiography was performed by a second observer for the same 30 patients again after the first echocardiography to assess the interobserver variability. Intraobserver and interobserver variability for lateral wall MPI were 0.04 and 0.06, respectively.

Statistical Analysis:

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS for Windows) software (version 13.0; SPSS Inc., Chicago, IL, USA). Differences in dichotomous variables were tested by the nonparametric chi-square test. Unpaired *t*-tests were used for between-group comparisons. Multivariate logistic regression analysis was performed to determine independent predictors of impaired MPI, defined as MPI $\geq$ 0.60. Gensini score, pre PCI TIMI flow, post PCI TIMI flow, duration from onset of angina to admission time, IRA, age, smoking status, hypertension, body mass index (BMI), and peak CK-MB variables were entered into the logistic regression model and backward elimination was applied. Removal criteria were defined as probability of stepwise above 0.10. The variables were expressed as mean ± standard deviation and P-values <0.05 were considered statistically significant.

Results:

Table I shows the characteristics of the study population. There were no significant differences in terms of age, cardiovascular risk factors, and BMI. Hemodynamic data at the time of admission were also similar. Biochemical and hemotological parameters like serum creatinine, white blood cell, peak CK-MB, and troponin I levels were comparable between 2 groups (Table I). The localization of MI, IRA, post PCI TIMI flow, Gensini scores, and other clinical factors were also similar (P > 0.05).

On conventional 2D echocardiographic examination, there were no significant differences in LV dimensions and EF in terms of LV functions (Table II). In contrast, wall-motion score index was higher in nonsmokers than smokers. In addition, the difference of TDI derived LV MPI between 2 groups reached statistical significance (Fig. 1). The difference in MPI originated from longer ET and shorter IVRT in smokers compared with nonsmokers. The ICT values were comparable in the 2 groups. According to the IRA, LV MPI was compared in the 2 groups (Fig. 2). When left anterior descending (LAD) and circumflex (Cx) arteries were the culprit, LV MPI was higher in nonsmokers than smokers. However, the right coronary artery (RCA) lesions were not associated with a difference in LV MPI values between the groups (Fig. 2). Logistic regression analysis was performed to determine the independent predictors of poor MPI (>0.60). The above-mentioned variables were entered in a stepwise logistic regression model. The LAD occlusion, nonsmoking status, age, and Gensini score were independent predictors of severe MPI impairment (Table III). Diastolic filling parameters were also analyzed. The E/A ratio and TDI derived Em/Am ratio were slightly lower in nonsmokers. Other echocardiographic findings are summarized in Table II.

Discussion:

This study firstly assessed LV MPI of smoker patients compared with nonsmokers during a first acute MI, which revascularized successfully with primary PCI. Although the patient characteristics such as extent of CAD, time from onset of chest pain to balloon dilatation, post procedural TIMI flow, and frame were similar in both patient groups, TDI derived LV MPI was preserved in smokers. We think that this could be associated with the smoker's paradox.

The MPI is a measure of combined systolic and diastolic myocardial performance.[24] The MPI is calculated by the following formula: (ICT + IRT)/ET.[25] In this formula, the ratio of ICT/ET represents systolic function.[12] An impaired

TABLE II

Comparison of Echocardiographic Measurements of the Groups

	Nonsmoker (n = 104)	Smoker (n = 325)	P
Two-dimensional echocardiography			
LVESD (cm)	3.4 ± 0.5	3.5 ± 0.6	NS
LVEDD (cm)	5.0 ± 0.4	5.0 ± 0.5	NS
LV ejection fraction (%)	52.6 ± 11.2	53.2 ± 9.9	NS
Wall-motion score index	24.2 ± 4.8	27.8 ± 6.9	0.03
LA (cm)	3.8 ± 0.4	3.7 ± 0.4	NS
Doppler echocardiography			
E (cm/sec)	66.7 ± 16.9	66.2 ± 17.1	NS
A (cm/sec)	68.6 ± 18.9	62.4 ± 17.1	0.01
E/A ratio	1.0 ± 0.4	1.1 ± 0.4	NS
EDT (msec)	166.8 ± 54.4	164.7 ± 48.8	NS
PAP (mmHg)	29.6 ± 7.5	29.4 ± 6.8	NS
Tissue Doppler echocardiography			
LV mean MPI	0.66 ± 0.14	0.59 ± 0.15	0.01
Lateral wall MPI	0.64 ± 0.18	0.58 ± 0.16	0.02
IVS MPI	0.67 ± 0.17	0.61 ± 0.21	0.01
Anterior wall MPI	0.65 ± 0.17	0.61 ± 0.17	0.04
Inferior Wall MPI	0.68 ± 0.18	0.60 ± 0.24	0.005
LV mean ICT (msec)	74.9 ± 17.6	71.2 ± 19.8	NS
LV mean IRT (msec)	91.2 ± 17.1	83.2 ± 17.9	0.001
LV mean ET (msec)	252.7 ± 29.7	263.7 ± 28.7	0.003
LV IRT/ET ratio	0.37 ± 0.08	0.32 ± 0.07	0.001
LV mean Sm (cm/sec)	7.74 ± 1.50	7.69 ± 1.66	NS
LV mean Em (cm/sec)	7.78 ± 3.70	7.90 ± 2.19	NS
LV mean Am (cm/sec)	10.40 ± 2.18	9.86 ± 3.37	NS
LV E/Em	9.22 ± 2.81	9.09 ± 3.56	NS
LV Em/Am	0.75 ± 0.31	0.84 ± 0.28	0.01

LVESD = left ventricle end-systolic diameter; LVEDD = left ventricle end-diastolic diameter; LV = left ventricle; LA = left atrial diameter; E = the peak mitral valve flow velocity during the early rapid filling phase; A = the peak mitral valve flow velocity during atrial contraction; E/A = ratio of peak velocity of early rapid filling wave to peak flow velocity at atrial contraction; EDT = deceleration time of early phase of mitral valve flow; PAP = pulmonary arterial pressure; MPI = myocardial performance index; IVS = interventricular septum; ICT = isovolumetric contraction time; IRT = isovolumetric relaxation time; ET = ejection time; Sm = systolic velocity; Em = early diastolic velocity; Am = late diastolic velocity.

systolic function is characterized by prolongation of ICT and shortening of ET period. Consequently, ICT/ET ratio increases in systolic dysfunction. On the other hand, the IRT is a measure of diastolic function, and it is prolonged in diastolic dysfunction. It has reported that the MPI was rapidly increased in the early phase of MI and the degree of the increase was associated with both mortality and morbidity.[26] In this study, we have demonstrated that both the ICT/ET ratio and IRT were more prolonged in nonsmokers compared with smokers resulted with a higher MPI. This is the major result of this study.

Reperfusion and Smoking:

Successful early reperfusion or revascularization of culprit coronary lesion limits the infarct size and consequently preserves LV function and improves the MPI.[27,28] Although smoking is considered to be the major modifiable risk factor for CAD, smokers have shown to have a better outcome after an acute MI in some former studies.[6–11,29] A systematic review of the previous studies showing beneficial effects of smoking in the setting of acute MI have demonstrated that this paradox was not evident when confounding clinical variables were taken into consideration.[30] In a recently published article, Chen et al.[31] reported favorable outcomes in younger (<45 years of age) smokers with acute MI. Similar results were obtained in patients with other forms of acute coronary syndromes that could not explained by younger age.[32] So, the controversy goes on. This study was conducted to evaluate potential mechanisms underlying the smoker's paradox. To assess the relationship between LV function and smoking in patients with acute MI, reviewing data on the effects of smoking on reperfusion is essential. Several studies addressed the better outcomes in smokers after fibrinolytic therapy, such as better patency of IRA, higher TIMI flow grade, better myocardial

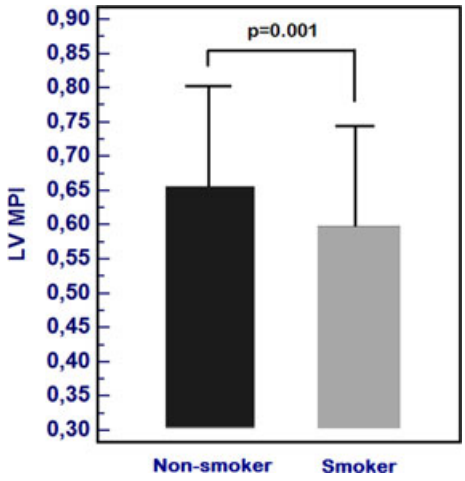

Figure 1. LV myocardial performance index of the groups.

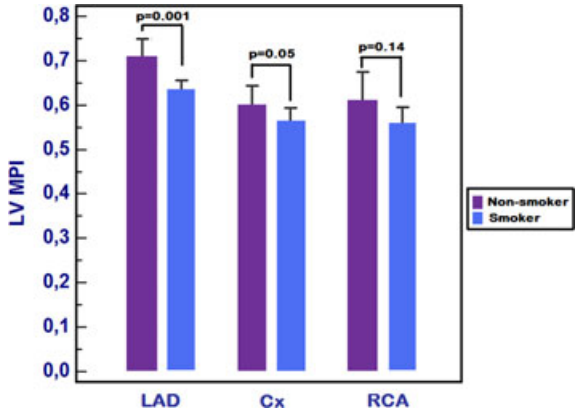

Figure 2. Mean LV myocardial performance index of 2 groups according to the infarct related artery.

perfusion grade, brighter myocardial blush intensity, and a faster rate of increase in blush intensity.[33,34] Weisz et al.[7] reported that TIMI-3 flow at baseline and post PCI was slightly more common in current smokers than nonsmokers but the frequency of TIMI 0, I and II flows were similar. The Primary Angioplasty in Myocardial Infarction study showed that thrombolysis or PCI had similar efficiency in smokers, but PCI was better than fibrinolytic therapy in nonsmokers.[35] Association between smoking status and procedural success rate remained controversial.[36] In this study, most of the patients underwent successful PCI, and both pre and post PCI TIMI flow were comparable in 2 groups.

MPI and IRA:

Previous studies showed that LV MPI was significantly increased in patients with anterior MI compared with inferior MI.[26,37,38] It was explained by the fact that patients with anterior MI had more extensive infarction and greater myocardial systolic and diastolic dysfunction.[38] In this study, the frequency of LAD to be the IRA was 50% for nonsmokers and 48% for smokers. The LV MPI increased in patients whose IRA was LAD compared with the patients whose IRAs were Cx and RCA. However, LV MPI of nonsmokers increased when both LAD and Cx were identified as IRA. In the multivariate logistic regression model, LAD as IRA was detected as an independent predictor of poor MPI (OR: 3.15; CI:1.68–5.91; P = 0.001). Thus, IRA and smoking status were 2 major independent predictors of mean LV MPI in this study. Several studies have reported that nonanterior MI was more frequent in smokers compared with nonsmokers.[7,9,39] However, the current study was unable to show the association between smoking status and IRA/MI type. The majority of previous studies included ex-smoker patients as nonsmokers. We thought that participation of patients with a history of smoking could create bias. For this reason, ex-smokers were excluded from this study.

Mean duration of onset of chest pain to reperfusion was about 4 hours, and the mean difference between 2 groups was only 10 minutes (P > 0.05). Despite relatively short reperfusion time and higher TIMI flow in IRA, the percent of poor MPI was high in both nonsmokers and smokers (66.0% vs. 44.7%). We suggested that the restoration of epicardial coronary flow with early invasive strategy has found to be not sufficient for preservation of myocardial performance. The possible underlying mechanisms of this result could be poor microvascular perfusion and ischemia reperfusion damage. This study did not assess myocardial blushing as a marker of myocardial perfusion, so we did not have data to interpret the microvascular function. The consistent findings obtained by various animal models of cardiac ischemia reperfusion injury suggested that hypoxia increased myocardial tolerance to ischemia by activating protective mechanisms.[40,41] Chronic hypoxia, increased oxidative stress, and deficient antioxidant activity as a result of long-term cigarette smoking were associated with an imbalance in myocardial oxygen demand and disturbances of the energy-consuming process of ventricular diastolic functions.[42–45] We speculated that chronic hypoxia may provide ischemic preconditioning in the early phase of coronary occlusion. Less impaired LV MPI in smokers with acute MI due to chronic exposure to hypoxia could be a possible explanation for "the smoker's paradox."

TABLE III

Multivariate Logistic Regression Analysis for the Predictors of Poor MPI (>0.60)

	β	Standart Error	P	OR	95% CI
First step					
Nonsmoker	1.078	0.560	0.054	2.940	0.98–5.83
BMI	0.118	0.038	0.18	1.05	0.84–1.21
Gensini score	0.006	0.003	0.14	1.006	0.99–1.010
IRA (for LAD)	1.162	0.314	0.001	3.196	1.73–5.91
Post PCI TIMI flow	2.025	1.209	0.09	7.57	0.71–10.82
Time of symptom onset to revascularization	−0.134	0.083	0.10	0.87	0.74–1.20
Age	0.112	0.044	0.01	1.12	1.03–1.22
Hypertension	−1.341	.825	0.10	0.26	0.05–1.32
Peak CK-MB	0.001	0.002	0.62	1.001	0.99–1.01
Last step					
Nonsmoker status	1.014	0.282	0.01	2.756	1.59–4.87
IRA (for LAD)	1.148	0.321	0.001	3.153	1.68–5.91
Gensini score	0.008	0.004	0.049	1.07	1.01–1.14
Age	0.114	0.044	0.01	1.12	1.03–1.22

Dependent variable = MPI $\geq$ 0.60; Nagelkerke R^2 = 0.32 Model statistic P = 0.01.
MPI = myocardial performance index; BMI = body mass index; IRA = infarct related artery; LAD = left anterior descending artery; PCI = percutaneous coronary intervention; TIMI = thrombolysis in myocardial infarction; CK-MB = creatine kinase myocardial band.

This study firstly indicated that LV dysfunction assessed with the tissue Doppler echocardiographic MPI is more prominent in nonsmoker young males after a first acute MI. The clinical significance of this finding is that assessment of MPI could identify patients with an increased risk of adverse outcome especially in nonsmoker patients. The results of this study should not be interpreted as paradoxically beneficial effect of cigarette smoking. It remains very clear that cigarette smoking is the chief, avoidable cause of CAD and death in society, and the most important public health issue. Physicians should be wise to see the elephant in the living room and encourage the patients to quit or reduce the intensity of smoking. It should be offered to smokers during hospitalization, at every office, or clinic visit. Patients who stop or reduce intensity of smoking after an acute MI had fewer adverse events and an improved survival.[46,47]

Smoking and Other Echocardiographic Parameters:

The ratio between transmitral early diastolic velocity determined by conventional Doppler echocardiography and early diastolic mitral annular velocity assessed by TDI was shown to be a reliable index of LV filling pressure in the presence of diastolic dysfunction.[23] This index was similar in both groups. However, the ratio of E/A and TDI derived Em/Am were slightly decreased in nonsmokers as a result of LV abnormal relaxation. A slight impairment of diastolic function in smokers was contributed to the abnormal MPI in these patients.

Study Limitations:

The primary limitation of the study was strict exclusion criteria. In this study, the effect of long-term smoking was assessed in a homogeneous sample of middle-aged (40–65 years) and nondiabetic males. Previous reports revealed that LV MPI is impaired due to aging in a linear manner.[48] Aging is also related to a higher prevalence of comorbid conditions, such as hypertension, diabetes mellitus, and diastolic dysfunction. All of these comorbid conditions impair the LV MPI.[49] Also, smokers develop MI a decade earlier than nonsmokers, which is an obvious bias in favor of smokers.[5] Taken together, we thought that aging could have a strong effect on LV MPI. For this reason, we studied a relatively younger population to minimize the effect of possible age difference on LV MPI between smokers and nonsmokers. We thought that an age-adjusted statistical model may not be enough to suppress effects of age on LV MPI. Secondly, diabetic subjects were excluded from the study. A previous study reported that diabetes mellitus is associated with impaired MPI without CAD,[50] extensive CAD, and frequent thrombotic complication during revascularization.[51,52] On the other hand, diabetics have substantially lower smoking percentage compared with nondiabetic subjects. Diabetic subjects could cause bias in favor of smokers.

For this reason, the patients with diabetes mellitus were excluded from the study. Furthermore, females were excluded due to negative effects of menopause and ovulation phases on LV MPI.[53] Another major limitation was lack of myocardial blush grading (MBG). Longer angiographic records and optimal opaque injection are required to obtain reliable angiographic views to assess MBG. In this study, only 52% of angiographic recordings were suitable for MBG. This was associated with avoidance of redundant contrast injection during acute MI. As a result, we did not assess myocardial blush scores. The current study was not designed to investigate mortality and major adverse cardiac events and instead focused on effects of smoking in patients with MI who were examined echocardiographically.

Conclusions:

This study showed that chronic smokers have less deteriorated left ventricular function after an acute MI as assessed by TDI techniques. After the reperfusion procedure, preserved MPI in smokers could be the cause of "the smoker's paradox." Like nonsmokers, smokers still require close monitoring for adverse outcomes despite their relatively lower post infarct MPI. Smokers who stop or reduce intensity of smoking after an acute MI had fewer adverse events and an improved survival, so they are encouraged to quit smoking in every medical contact.

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