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Changes in smoking behavior and adherence to preventive guidelines among smokers after a heart attack

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Abstract

Objective Risk factor modification is key to preventing subsequent cardiac events after a heart attack. This study was designed to investigate the disparity between preventive guidelines and clinical practice among smoking patients. **Methods** The study was carried out in smokers admitted with myocardial infarction (MI). A total of 275 patients who had been regularly followed for over one year after MI were randomly selected and enrolled in this study. We investigated changes in smoking behavior and the adherence rate to ACC/AHA Guidelines for secondary prevention in patients with coronary artery disease at the time of, and one year after, the index event. **Results** The study population consisted of 275 patients (97.1% males) with a mean age of 57.0 ± 11.2 years. Achievement of target goals at one year was as follows: smoking cessation, 52.3%; blood pressure, 83.9%; HbA1c, 32.7%; lipid profile, 65.5%; and body mass index (BMI), 50.6%. Over one year, 80% of the patients attempted to quit smoking; 27% of them re-started smoking within one month after discharge while 65% succeeded in cessation of smoking. At one year, only 52% of the patients overall had stopped smoking. From the multivariate logistic analysis including smoking patterns and clinical characteristics, the severity of coronary artery disease was the only independent predictor for smoking cessation (Relative risk (RR): 1.230; $P = 0.022$). **Conclusions** Only a small percentage of MI patients adhere to guidelines for secondary prevention and a sizable proportion fail to stop smoking. These findings underscore the need for an effective patient education system.

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Keywords: Coronary artery disease; Myocardial infarction; Prevention; Smoking

1 Introduction

It is well documented that risk factors management is the key to preventing recurrent events and improving survival of patients with established coronary artery disease (CAD). These include smoking cessation, blood pressure control, regulation of blood glucose in diabetes patients, lipid management, and weight reduction. Smoking cessation, in particular, is emphasized as well as avoidance of exposure to second hand tobacco smoke. Among cigarette smokers, increased hematocrit, fibrinogen and thrombin generation and enhanced platelet activation induce a hyper-coagulable and proinflammatory state which can lead to coronary artery disease. Quitting smoking is associated with risk reduction of all-cause mortality among patients with coronary artery disease,[1] and one study reported that patients who continue to smoke after myocardial infarction (MI) have a 50% higher risk of recurrent coronary events compared to non-smokers.[2] Despite the well known advantages of smoking cessation in CAD and physicians' efforts to encourage smoking cessation, many patients continuing to smoke after a diagnosis of CAD. Other risk factors are more easily controlled since they are much more dependent on medication

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use than patient's behavior. The aim of this study was to investigate the gap between preventive guidelines and clinical practices among smoking patients.

2 Methods

2.1 Patients

The study involved smokers who were admitted with MI at five major cardiac centers (Yeungnam University Hospital, Chonnam National University Hospital, Chungbuk National University Hospital, Kyunghee University Hospital, and Hanrim University Hospital) in South Korea. From May 1999 to May 2008, a total of 275 smoking patients who were regularly followed for over one year after MI were randomly selected and enrolled in this study.

2.2 Outcome measures

We investigated changes in smoking behavior and adherence rate to the ACC/AHA Guidelines for secondary prevention in patients with CAD at the time of, and one year, after heart attack. Smoking patterns, blood pressure, HbA1c, lipid profile, body mass index and one year major adverse cardiac events (MACE) were analyzed. Based on ACC/AHA Guidelines,[3] we set goals for secondary prevention as follows: complete cessation of smoking; target blood pressure < 140/90 mmHg, < 130/80 mmHg if the patient had diabetes or chronic kidney disease; in diabetes patients, goal of HbA1c was < 7%; for lipid management, low-density lipoprotein cholesterol (LDL-C) < 100 mg/dL and non-HDL-C (non-high-density lipoprotein cholesterol) < 130 mg/dL; and/or weight management with a body mass index between 18.5 and 24.9 kg/m^{2}as goal. Information about smoking habits was obtained from the patient or a representative at the time of admission. The severity of CAD was estimated by the number of vessels with severe stenosis.

2.3 Data analysis

The results are expressed as mean ± SD for continuous variables, or numbers and percentages for categorical variables. Comparisons of categorical and continuous variables were done using the Chi-square test and the Student's *t* test, respectively. Relative risk and 95% confidence interval for predictors for smoking cessation and risk of cardiac events was estimated by use of multivariate logistic regression analysis. In all analyses, statistical significance was accepted at $P < 0.05$. Data were analyzed with the use of SPSS 12.0 for Windows software (Chicago, IL, USA).

3 Results

3.1 Baseline characteristics of the patients

The baseline characteristics of the studied patients at the time of presentation with heart attack are presented in Table 1. The mean age was 57.0 ± 11.2 years with male predominance (97.1%). Among the 275 patients: 55 (20.0%) had diabetes; 95 (34.5%) had hypertension; and 98 (35.6%) had dyslipidemia. ST-segment elevated myocardial infarction (STEMI) was diagnosed in 57.1% of patients. The prevalence of previous MI (6 patients, 2.2%) and previous intervention (4 patients, 1.5%) was low. The majority of patients (54.9%) underwent drug-eluting stent implantation during percutaneous coronary intervention (PCI). The mean left ventricular ejection fraction was 53.1 ± 10.7% and peak creatinine kinase (CK-MB) was 173.1 ± 205.3 mg/dL.

Table 1. Baseline demographics of the patients studied (*n* = 275).

Age (yrs)	57.0 ± 11.2
Men	267 (97.1%)
Diabetes	55 (20.0%)
Hypertension	95 (34.5%)
Dyslipidemia	98 (35.6%)
Previous history	
CVA	9 (3.3%)
Myocardial infarction	6 (2.2%)
Coronary intervention	4 (1.5%)
CABG	0
LVEF, %	53.1 ± 10.7
Peak CK-MB, mg/dL	173.1 ± 205.3
Diagnosis	
STEMI	157 (57.1%)
NSTEMI	118 (42.9%)
Coronary intervention	
Balloon angioplasty	15 (5.5%)
BMS implantation	28 (10.2%)
DES implantation	151 (54.9%)

Data are presented as *n* (%). BMS: bare metal stent; CABG: coronary artery bypass surgery; CK-MB: creatine kinase-MB; CVA: cerebrovascular accident; DES: drug-eluting stent; LVEF: left ventricular ejection fraction; NSTEMI: non-ST-segment elevated myocardial infarction; STEMI: ST-segment elevated myocardial infarction.

3.2 Adherence to guidelines for secondary prevention of coronary artery disease

Table 2 summarizes changes in blood pressure, body mass index (BMI), abdominal circumference, HbA1c and lipid profile at the time of the index event and at one year follow up. Diastolic blood pressure was lower, BMI was higher while abdominal circumference did not change significantly at one year after the index event than at baseline.

In diabetes patients, HbA1c level was lower at one year reflecting better glycemic control. In terms of lipid profile, total cholesterol and LDL-C were significantly lower at one year *vs*. baseline. Based on ACC/AHA Guidelines, the following proportion of patients achieving the target goals at baseline: 0% for smoking cessation, 70.5% for blood pressure, 5.4% for HbA1c level in diabetes patients, 24.5% for LDL-C level, and 61.6% for BMI at the time of index event. At one year after the event, the proportions of patients achieving the target goals were as follows: 52.3% for smoking cessation, 83.9% for blood pressure, 32.7% for HbA1c level in diabetes patients, 65.5% for LDL-C level, and 50.6% for body mass index (Figure 1).

Table 2. Changes in risk factors after one year.

Risk factors	Baseline	One year	*P*-value
Systolic pressure, mmHg	120.1 ± 22.5	123 ± 13.4	0.069
Diastolic pressure, mmHg	75.1 ± 14.5	72.3 ± 13.4	0.005
BMI, kg/m^2	24.2 ± 3.2	25.0 ± 3.3	< 0.001
Abdominal circumference, cm	87.8 ± 8.1	88.6 ± 8.3	0.051
HbA1c, %	7.2 ± 1.9	6.7 ± 1.5	0.004
Total cholesterol, mg/dL	193.1 ± 41.8	160.6 ± 38.8	< 0.001
LDL-C, mg/dL	124.4 ± 35.7	91.1 ± 32.7	< 0.001
HDL-C, mg/dL	46.8 ± 12.8	47.3 ± 12.4	0.513
Triglyceride, mg/dL	143.9 ± 87.5	146.1 ± 95.8	0.696
Non-HDL cholesterol, mg/dL	146.9 ± 39.6	113.6 ± 38.2	< 0.001

BMI: body mass index; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol.

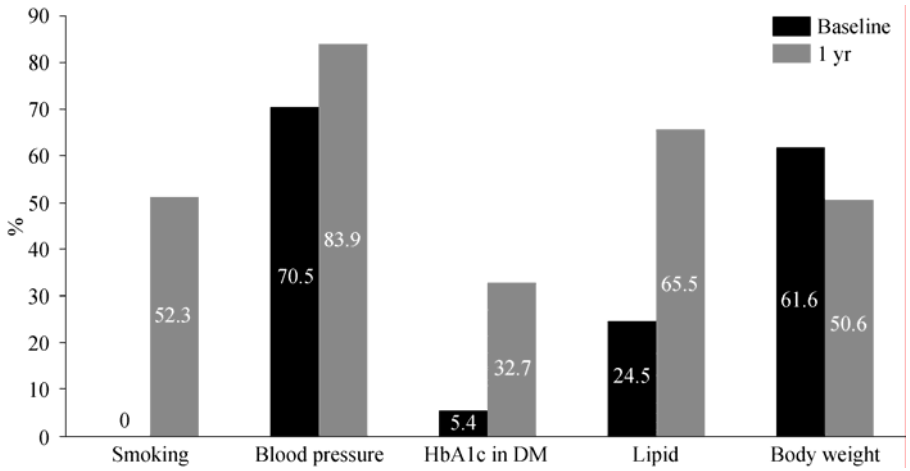

Figure 1. Percent of patients reaching target ACC/AHA Guideline. Percent of patients who reached target goals at the time of myocardial infarction and at one year after the index event.

3.3 Change of smoking habits

We analyzed the smoking habits of patients in detail. At one year after the index event, 52.3% of patients had ceased smoking, and 30.6% of patients decreased the amount of smoking. However, in 16.0% of patients there was no change in the smoking pattern, and 1.1% of patients smoked more than before the index event (Figure 2). Among 131 patients who failed to cease smoking, the average time to restart smoking after discharge was 4 ± 7.3 months; many (59 patients, 45%) restarted smoking within one month after discharge. Among 131 current smokers still smoking at one year post index event, 77 patients (58.8%) tried to quit smoking, but failed (Table 3).

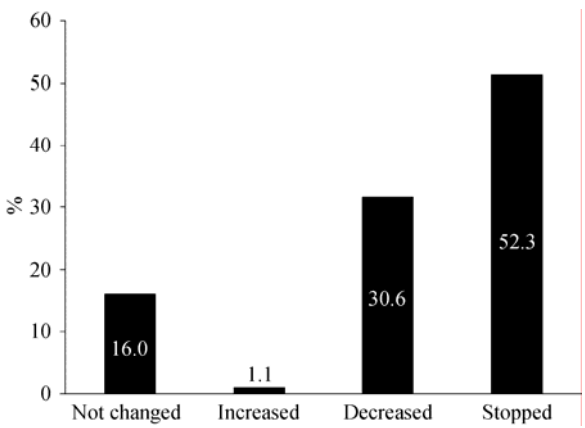

Figure 2. Changes in smoking habits at one year. Smoking habits of patients at one year after index event. In 16.0% of patients, smoking pattern was unchanged, 1.1% of patients smoked more than before index event, 30.6% of patients decreased smoking, and 52.3% of patients stopped smoking.

3.4 Predictor for smoking cessation and relation to coronary event

There was no previous smoking pattern (smoking onset age, amount and duration) associated with the success of stop smoking. From the multivariate logistic analysis, including smoking patterns and clinical characteristics, the severity of coronary artery disease was the only independent

predictor associated with smoking cessation (relative risk (RR): 1.230, 95% CI: 0.864–1.752, P = 0.022) (Table 4). On the other hand, continuous smoking was not an independent predictor of re-intervention, or MI at one year (Table 5).

Table 3. Trial and failure of smoking cessation (n = 131).

	Current smoker
Tried to quit smoking	77 (58.8%)
Time to re-smoke after discharge, months	4.0 ± 7.3
< 1 month	59 (45%)
1–6 months	44 (33.6%)
6–12 months	24 (18.3%)
>12 months	4 (3.1%)
Amount, pack/day	0.7 ± 0.5

Table 4. Independent predictors for smoking cessation.

	RR (95% CI)	*P* value
Age (per 1 year old)	1.054 (0.992–1.119)	0.090
Onset of smoking (per 1 year old)	0.986 (0.921–1.056)	0.692
Amount of smoking (per 1 pack/day)	1.055 (0.628–1.773)	0.840
Duration of smoking (per 1 year)	0.962 (0.909–1019)	0.186
Drinking (per 1 time/week)	1.114 (0.860–1.445)	0.413
BMI < 24.9 (< 24.9 *vs.* ≥ 24.9), kg/m^2	1.411 (0.764–2.606)	0.272
CAD severity (per 1 lesion)	1.230 (0.864–1.752)	0.022

BMI: body mass index; CAD: coronary artery disease; CI: confidence interval; RR: relative risk.

Table 5. Independent predictors for re-intervention or myocardial infarction.

	RR (95% CI)	*P* value
Stop smoking	2.858 (0.247–33.037)	0.400
BMI < 24.9 kg	0.601 (0.051–7.034)	0.685
LDL-cholesterol < 100 mg/dL	0.588 (0.034–10.329)	0.717
BP <140/90 or <130/80 mmHg	0.418 (0.122–1.425)	0.163
HbA1c <7% in diabetic patients	0.256 (0.029–2.280)	0.222
CAD severity	0.907 (0.597–1.379)	0.648
Therapy (Intervention or medical)	0.536 (0.121–2.382)	0.413

BMI: body mass index; BP: blood pressure; CAD: coronary artery disease; CI: confidence interval; RR: relative risk.

4 Discussion

Smoking is an established, and the only totally preventable, risk factor for CAD. Guidelines strongly encourage patients and their families to stop smoking and to avoid second hand smoke.[3] Many people stop smoking during their hospitalization for the ischemic episode. However, many of them relapse within a short time.[4] Our study also shows that many patients attempt to stop smoking after MI, however, only half of them succeed. It is well known that smoking after an MI incident is associated with an increased risk for recurrent coronary events, however, most smokers admitted to the hospital for acute coronary syndrome receive no smoking cessation support after discharge.

Smoking, as has been demonstrated, is associated with CAD, with the relative risk or odds ratio estimated between 1.5 to 3, or higher.[5,6] The exact mechanisms involved in cigarette smoking related cardiovascular dysfunction are unknown, but increased inflammation, thrombosis, and oxidation of LDL-C are potential mechanisms for initiating cardiovascular dysfunction.[7] These effects seem to be reversible, and significant reductions in mortality have been observed in patients with established cardiovascular disease who stopped smoking.[8] Accordingly, smoking cessation plays an important role in secondary prevention of coronary artery disease.

It has been reported that quitting smoking is associated with a reduced risk of total mortality with a pooled RR of 0.64 (95% CI: 0.58–0.71).[1] Also, smoking cessation is associated with a reduced incidence of second cardiac events, including cardiac death and non-fatal MI in patients with a history of MI.[9] Despite this knowledge, up to 20% of patients return to the habit after discharge and unsuccessful efforts at smoking cessation. Many physicians have tried to create effective ways to stop habitual smoking. A comparison study between smokers with intensive intervention (including regular meetings with counselors after discharge and individualized adjuvant pharmacotherapy) and with usual care shows that intensive smoking cessation intervention is not only effective in achieving cessation, but also reduces hospitalizations and total mortality. Thus, structured smoking cessation intervention administered to hospitalized smokers with cardiovascular disease should be considered in high-risk patients.[10]

Despite the advantage of smoking cessation in the context of acute MI, some studies have found that the mortality rate of smokers after acute MI may be lower than in non-smokers, what is termed a smoker's paradox. This phenomenon can be explained by individual differences in baseline characteristics and not by smoking status itself. Cigarette smokers, suffering an acute MI, tend to be younger with less diffuse coronary artery disease and fewer co-morbidities compared to non-smokers. Therefore, the role of cigarette smoking in the short-term prognosis after acute MI remains uncertain,[11] and the failure in our study to document a beneficial effect for smoking cessation could be secondary to the short observation period.

Smoking cessation was independently associated with a lower risk of MACE and recurrent angina. Furthermore, quitters comply more closely with medication schedules and the recommended lifestyle changes than do persistent smokers, who also benefit from smoking cessation in terms of long-term mortality.[12] According to a study,[13] the half of quitters had a weight gain of less than 1.5 kg by one year follow-up after PCI and weight gain could curtail the benefits from smoking cessation. However, after adjusting for weight gain and other factors, smoking cessation was independently associated with a lower risk of MACE and recurrent angina. Thus, although smoking cessation is recommended after PCI, weight control should also be highly encouraged for these patients.

This study has some limitations. First, it was a retrospective analysis. Second, the number of patients in the study population was relatively small. As such, the number of predictors of failure to quit smoking that could be analyzed was limited to < 7. We did not analyze important predictors, such as nicotine replacement therapy, the impact of smoking habits of family or partner of the patients, and education level. Third, follow-up duration was short to evaluate the full effects of smoking cessation. Fourth, information on smoking status was derived from self-reporting and not validated by nicotine measurements.

In conclusion, smoking cessation after an MI event (occurrence) reduced the incidence of recurrent coronary events as being the main determinant of reduced mortality. However, a sizable proportion of patients failed to stop smoking which underscores the need for aggressive management, active promotion, and effective education to improve the success rate of smoking cessation among patients with MI.[14]

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