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Environmental Tobacco Smoke and the Risk of Coronary Heart Disease

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Abstract

Background: Passive or environmental smoking is one of the important health problems, to which many adults and children are exposed in involuntary status.

Methods: In this hospital based case-control study, household exposure to tobacco smoke as an estimate of passive smokers was conducted in Hamadan, western Iran. The purpose was to find the relationship between environmental smoking and incidence of coronary heart disease (CHD). We interviewed 69 patients with CHD and 69 without it as control group. The confounded factors (hypertension, hypercholestrolemia, diabetic mellitus, type a personality, and familial history) were removed as exclusion criteria while suppliers of oral contraceptives, physical activity, martial status and age (±3) were matched.

Results: The findings showed that 54% of patients and 48% of the controls were exposed to tobacco smoke. The odds ratio of incidence of CHD was 1.26 and confidence interval (CI) as 0. 65-2. 46.

Conclusion: There was no significant relationship between passive smoking and CHD. More study on large sample is needed to assess environmental smoking effect on CHD.

Keywords: *Passive smoking, environmental tobacco smoke, coronary heart disease, tobacco*

Introduction

Smoking is one of the important behavioral risk factors for coronary heart disease (CHD), lung disorders, and cancers, known as a major preventable risk factor of coronary heart disease (CHD) (1-3). Smoking causes about 17% of mortality in over 65 yr age group and is responsible for incidence of 23% of all the CHD in under 45 yr age group (4). It also increases CHD risk 2 to 3 times among smokers versus non-smokers (1, 3). Side effects of tobacco on coronary arteries include the formation of arterial plaque due to carbon monoxide (CO) and the effect of nicotine on the adrenergic secretion, which not only causes hypertension but also increases low-density lipoproteins (LDL) oxidation. In other words, nicotine reduces the role of HDL for preventing CHD (1, 5). In addition, nicotine causes platelets aggregation and consequently changes level of fibrinogen in the serum. Therefore, all of these pathways could cause CHD (2). Tobacco, also, affects the non-smoker people who are exposed to the environmental tobacco smoke (ETS) as passive smokers. In fact, ETS increases the risk of CHD in a prolonged time (5-8). Involuntary exposure to tobacco occurs when a non-smoker person is located or lives in an indoor environment for a long time (years) where a smoker is living with him/her. Tobacco smoke consists of nitrogen oxide, nicotine, CO, carcinogens, and co-carcinogens. The undiluted tobacco smoke has

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high pH, small particles, and high density of CO, carcinogens, co-carcinogens, and gas-phased constituents (5).

In indoor situation, these components are decreased by only 15%. Therefore, in the room airflow there would be 85% of undiluted tobacco smoke components and density (9). Many research studies have been carried out about ETS and the incidence of CHD, which have reported side effects of ETS on pregnancy, CHD, cancers, and pulmonary disease (10, 11).

In accordance with the abovementioned background, the purpose of the present study was to assess the effect of ETS on CHD among household women who had prolonged time smoker husband in Hamadan, Iran.

Materials and Methods

This study was an analytic case-control study carried out in Hamadan (Iran) on non-smoker married women who had prolonged smoker husbands during 1998-2000. In this study, 69 non-smoker CHD patients were compared with 69 non-smoker control patients hospitalized in the hospitals of Hamadan University of Medical Sciences for diagnostic and treatment procedures.

Patient (case) group were women hospitalised in the cardiac care units (CCU) and their CHD had been investigated by cardiologist based on clinical and laboratory study and electrocardiography (ECG). Patients having diabetic mellitus, hypertension, hypercholestrolemia, type a personality, and familial history were removed as exclusion criteria while suppliers of oral contraceptives, physical activity, martial status and age (±3) were matched. For each patient, one control sample was selected from the surgical or internal medicine wards whose age was±3 yr versus the selected case.

In this study, diagnosis of CHD was based on ECG, creatine phosphokinase (CPK), lactate dehydrogenase (LDH), patients' main complaint, and patients' medical history, assessed by cardiologist. Analysis was conducted using SPSS 9 and an alpha level of .05 for all statistical tests.

Results

Analysis of the baseline data showed that there was not significant difference between the case and control group according to age, oral contraceptive consumption, job status, family history, and body mass index (BMI). Therefore, both groups were matched with each other for risk factors of CHD. Hence, the probability of the bias occurrence seems to be in the lowest level (Table.1). As a result, this finding could indicate the risk of ETS on occurring CHD.

Rate of smoking by the husbands of CHD patients was estimated 11.74 cigarettes per day while it was equal to 0 9.04 cigarettes per day for the husbands of control group, the case that groups *t*-test did not show significant difference (*t*: 1.29, *P*: 0201) between case and control groups for rate of smoking per day. Moreover, the mean duration of ETS in patients group was 18.3 yr and in the control group as 16.23 yr. Smoking behavior of subjects is shown in Table 2.

The main finding of this study showed that 37 cases from 69 CHD patients and 33 cases from 69 control patients had been exposed by ETS involuntary, which showed an odds ratio of 1.26 (confidence interval :0. 65-2.46) for occurring CHD among case group. This result indicates that there is no significant relationship between CHD and ETS (Table 3). One of the considerable findings includes the existence of significant relationship between CHD and rate of pregnancy that groups *t*-test showed significant difference between the rate of pregnancy and occurring CHD (Table 4).

Table 1: Age, OCP consumption, job status, familial history, and BMI statistical indices among patients and control group

Variables		CHD Patients	Control	Test	*P*
OCP consumption (year)		$\bar{X}$ = (SD) 1.02(2.29)	$\bar{X}$ = (SD) 1.36(3.72)	*t* -0.65	0.516
Age (yr)		$\bar{X}$ = (SD) 66.23(9.55)	$\bar{X}$ = (SD) 66.4(12.74)	*t* -0.08	0.929
Job status	Household	65(94.2%)	66(95.7%)	X^2	0.698
	Employee	4(5.8%)	3(4.2%)	0.15	
Familial history	Yes	9(13%)	7(10.1%)	X^2	0.594
	No	60(87%)	62(89.9%)	0.28	
BMI	BMI<29.1	3(4.8%)	3(4.8%)		0.331
	26.1-29	10(14.5%)	13(19%)	X^2	
	18.1-26	46(66.1%)	36(52.4%)	3.42	
	BMI>18	110(4.5%)	17(23.8%)		

Table 2: Smoking behavior among husbands of patients and control groups

Number of cigarette group	Non-exposed	n>10	11-20	20-30	n<30
CHD patients (n=69)	34(49.3%)	3(4.3%)	21(30.4%)	6(8.7%)	5(7.2%)
Control(n=69)	36(52.2%)	7(10.1%)	16(23.1%)	9(13%)	1(1.4%)

X^2, d.f:4, P.value:0.231

Table 3: Incidence rate of CHD among exposed and non-exposed to ETS

Exposed Group	Positive	Negative
CHD Patients(n=69)	37 (54%)	32 (46%)
Control(n=69)	33 (48%)	36 (57%)

X^2, d.f: 1, P.value:0. 261
Odds ratio: 1.26(Confidence Interval: 0. 65-2.46)

Table 4: Mean of pregnancy rate among exposed and non-exposed to ETS

Pregnancy Rate Groups	Means	SD	*t*	*P*
CHD patients (n=69)	6.98	2.36	4.73	<0.001
Control (n=69)	4.82	2.97		

Discussion

The main goal of this study was to find the relationship between ETS and CHD among housewives who had prolonged smoker husbands. Survey on relationship between ETS and CHD among women has begun since 1980. Some studies indicated that CHD occurs among passive smokers more than among non-exposed people (4, 6, 9, 11, 13). Involuntary exposing to nicotine happens for many people. Therefore, a large part of population is exposed to ETS. As a result, studying about risk of ETS on tobacco related disease would be useful for designing protective legislations and rules.

It would be important and necessary to pay attention to epidemiological aspects of ETS and ETS consequences in planning primary prevention program, which could reduce diagnostic and treatment procedures' cost. Findings of this study indicated that the probability of CHD

among women who were exposed to ETS, is 0.26% more than the non-exposed to ETS. Another research carried out in Kerman, Iran estimated an odds ratio of 1.3 (CI: 1.2-1.4), which is similar to findings of this study (14). In addition, He et al. (7) reported an odds ratio of 1.24 for CHD among married women who had been exposed to ETS. Moreover, relative risk of CHD among passive smokers equal to 1.02 was reported in another study (15). Passive smoking is associated with a small increase in the risk of CHD. Given the high prevalence of cigarette smoking, the public health consequences of passive smoking with regard to CHD may be important (16). However, McElldufet al. reported odds ratio of 1.89 and 1.98 in Australia and New Zealand, respectively (9). However, most of the studies reported an odds ratio between1.2-1.4 (4, 5, 8, 11). It is also reassuring that the estimate was essentially the same when studies adjusting for some of the risk factors for CHD were pooled. However, other lifestyle factors such as diet and exercise will also play a part in an individual's risk

Although, this study and some other studies reported poor relationship between ETS and CHD, it is necessary to do more study for finding effect of ETS on CHD, fetus, neonates, lung disease, and cancers. Recognition of ETS effect on occurring disease would be important for designing primary preventative programs.

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