CIGARETTE SMOKING AND CARDIOVASCULAR DAMAGE: ANALYTIC REVIEW OF THE SUBJECT

A Leone

ABSTRACT

A large series of epidemiological, clinical and pathological studies relate cigarette smoking to the development of cardiac damage. Data of our studies on both active and passive smoking show the following results. (A) Active smoking: (i) clinical and electrocardiographic signs of myocardial ischaemia were seen in 266 (38%) out of 700 smokers vs 25 (12.5%) out of 200 non-smokers ($p < 0.01$) with similar characteristics, (ii) a significantly higher incidence ($p < 0.05$) of reinfarctions in 443 smokers who continued to smoke (200 reinfarctions, 45%) compared to 724 subjects with a previous infarction who stopped smoking (289 reinfarctions, 40%), (iii) a significantly higher incidence of reinfarction in smokers with a previous myocardial infarction who smoked more than 15 cigarettes per day compared to a similar population who smoked less than 15 cigarettes per day (118/212, 56% vs 82/231, 35%). (B) Passive smoking: 19 male volunteers (9 healthy and 10 with a previous myocardial infarction), who did not smoke, underwent exercise stress testing twice; once in a smoke-free environment and once in a smoking environment. Cardiac performance of these subjects was impaired significantly in the smoking environment. Pathologically, severe coronary alterations, especially in users of oral contraceptives, massive myocardial infarctions and focal myocardial lesions were seen. Several variables such as type of smoking, subject and environment may interfere with experimental results. When we standardise these variables, cardiac damage caused by cigarette smoking is an undoubted fact.

Keywords: *cigarette smoking, cardiovascular damage.*

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INTRODUCTION

It is a common knowledge that cigarette smoking is a potential hazard for daily life.

Different studies[1-31] showed the cardiovascular system as a target organ for cigarette smoking. Both active or passive exposure to smoking did damage to the heart. Moreover, consumption of cigarettes is decreasing in the developed countries while increasing in the developing ones. Table I shows yearly cigarette consumption per adult – people 15 years and over, both sexes – in Asian countries[32]. By analysis of these data we can deduce that cigarette consumption is a widespread habit in the above countries with a consequent risk for individual health.

The purpose of this review is to describe the typical alterations of cardiovascular system, discussing our previous studies on the subject.

Previous Studies

We began to study the problem of smoking/cardiovascular damage by examining ischaemic outpatients. Both active and passive smoking have been analysed in our papers.

ACTIVE SMOKING

Active cigarette smoking was examined in our first studies[9-16,18]. Myocardial ischaemia affected significantly 266 (38%) out of 700 smokers vs 25 (12.5%) out of 200 non-smokers with similar characteristics ($p < 0.01$).

Division of Medicine
City Hospital Pontremoli
Via Provinciale 27
19030 CASTELNUOVA MAGRA (SP)
Italy

A Leone, MD
Head

Table I – Yearly cigarette consumption per adult in some Asian countries (latest values available: years 1985-88).

Countries	Cigarettes Consumption (No./adult)	Countries	Cigarettes Consumption (No./adult)
Afghanistan	50	Bangladesh	270
China	1590	Hong Kong	1580
India	160	Indonesia	1050
Iran	620	Iraq	980
Israel	2310	Japan	3270
Jordan	1700	Korea	2660
Korea DPR	1180	Kuwait	2760
Lebanon	2880	Malaysia	1840
Nepal	150	Pakistan	660
Philippines	350	Saudi Arabia	2110
Singapore	2280	Sri Lanka	500
Syria	2050	Thailand	900
Turkey	1970	Vietnam	670

Leone et al[15] also analysed the long-term prognosis of 1,167 smokers who survived a first acute myocardial infarction. A significantly ($p < 0.05$) higher incidence of reinfarctions was seen in the 443 smokers, who continued to smoke after their infarction – 200 cases of reinfarction, 45%, compared to the 289/724 reinfarctions (40%) of those patients who stopped smoking after their first infarction. This report also studied the number of deaths which was similar for the two groups.

For smokers who survived a first acute myocardial infarction, greater mortality was also found in other studies[24-29], although differing in number with regard to mortality, reinfarction and smoking habit of the people studied. From the above observations, it can be concluded that death and reinfarction in smokers with a previous myocardial infarction are due to different mechanisms. Death is often caused by the infarction itself, while the reinfarctions could be a consequence of coronary atherosclerosis progression due to cigarette smoking. If such

a consideration is true, we would observe more reinfarctions in smokers with a previous myocardial infarction, who smoked a greater number of cigarettes. In confirmation of this hypothesis, a significantly higher incidence of reinfarctions was seen in those smokers who smoked more than 15 cigarettes per day after the infarction compared to patients who continued to smoke but less than 15 cigarettes per day[38]. Subjects were followed-up for a maximum period of 6.5 years.

Moreover, among ischaemic heart diseases acute myocardial infarction, reinfarction and chronic angina, which are characterised by a progression of coronary atherosclerosis, are more closely related to cigarette smoking than sudden death and coronary vasospasm[1], where functional disorders are frequently involved.

Necropsy studies[11,12,14,31] also showed more advanced lesions of coronary arteries and myocardium of the smoker when compared to nonsmoker, especially for those subjects in whom other risk factors were associated with smoking. Leone et al[14] observed more advanced coronary narrowings, higher incidence of occlusive thrombi and three-vessel coronary disease in 68 smokers, who died from acute myocardial infarction, when they were compared to 12 similar subjects, who were nonsmokers. Likewise, findings of Auerbach et al[31] showed more advanced narrowing of coronary arteries in the smokers. Cardiovascular diseases related to cigarette smoking as well as type of coronary and myocardial lesions are listed in the Tables II and III.

Table II – Main cardiovascular diseases related to cigarette smoking.

1. Ischaemic Heart Disease
2. Cerebrovascular Disease
3. Hypertension
4. Aortic Aneurysm
5. Peripheral Vascular Disease

Table III – Type of coronary and myocardinal lesions related to cigarette smoking.

Coronary lesions
- Advanced atherosclerosis
- Occlusive thrombi
- Three-vessel disease

Myocardial lesions
- Myocardial infarction
- Focal myocardial haemorrhage
- Experimental focal myocarditis (direct effect of carbon monoxide)

A separate remark is useful to clarify the relationship between cigarette smoking, oral and/or pathological contraception and ischaemic heart disease as our studies[11,12] seem to show. Use of oral contraceptives, a practice involving a large number of women in the developed countries, increases coronary heart disease risk. From age 30 onwards, absolute excess risks become sizeable, particularly when other major risk factors are present, especially cigarette consumption. Despite the overall safety of oral contraceptive agents, users are at risk for several serious side effects. Deep vein thrombosis and pulmonary embolism are disorders related frequently to administration of these drugs. The use of oral contraceptives is also associated with an increased risk of thromboembolism after surgery. Thromboembolic and haemorrhagic strokes have been demonstrated in users of oral contraceptives. When women were treated with oestrogens, no protection against the development of myocardial infarction occurred[11,12]. One of the two cases we described[11] was characterised by massive transmural myocardial infarction and severe coronary narrowing in a young woman (age: 35 years), who smoked 35-40 cigarettes per day and used oral contraceptives. This patient, who had exertional angina and silent myocardial ischaemia detected by Holter monitoring, was admitted to the Coronary Care Unit for unconsciousness due to ventricular fibrillation. After repeated direct-current cardioversions that restored sinus rhythm, acute myocardial infarction was seen in the electrocardiogram. The patient died four days later and post mortem examination showed massive transmural myocardial infarction involving about 35% of myocardial mass as well as severe coronary lesions with occlusive thrombi that affected the left anterior descending artery.

In conclusion, there is no doubt about the correlation of active smoking to diseases of the heart and blood vessels.

The mechanisms which are responsible for cardiovascular damage may also be defined: lesions of the blood vessels and myocardium mediated directly by carbon monoxide[14,17,23] and functional disorders mediated by nicotine[17,20,22].

PASSIVE SMOKING

Observations about passive smoking as a factor which causes cardiovascular impairment have appeared in the last decade.

Environmental tobacco smoke is an aged, diluted mixture of sidestream and exhaled mainstream smoke from combustion of tobacco products such as cigarettes, cigars and pipes. However, the effects of passive smoking from cigarettes are only beginning to be known.

That phase of smoke we define as sidestream smoke contains greater concentrations of many smoke constituents which are diluted in a large volume of air depending on indoor atmosphere. However, although diluted, pollutants are able to cause cardiovascular damage after either acute or chronic exposure. Chronic exposure to passive smoking may lead to myocardial infarction and cardiac failure[33], whereas acute exposure seems to impair cardiac performance during exercise stress testing[3,4].

Leone et al[3,4] studied the effects of passive smoking on the cardiovascular system of 19 non-smoking male volunteers. These consisted of 9 healthy subjects with a mean age of 30.5 ± 8.5 years and 10 patients who survived a first acute myocardial infarction, whose mean age was 53.8 ± 8.5 years. These people underwent exercise stress testing twice: once in a smoke-free environment and once in the same environment polluted by 30 – 35 ppm carbon monoxide concentration reached by a method of cigarette combustion described previously. In summary from this experiment we reached the following conclusion: acute exposure to passive smoking impaired cardiac performance of studied subjects. For survivors from acute myocardial infarction in a smoking environment, the following observations were made: the peak of exercise was reduced, time for recovery to pre-exercise heart rate was prolonged, post-exercise plasma carbon monoxide concentration was increased, and ventricular arrhythmia was displayed. Healthy people had

prolonged time for recovery to pre-exercise heart rate in the same environment. Therefore, both groups showed impaired cardiac performance in a smoking environment, although with different results.

In conclusion, passive smoking had been shown to impair cardiac performance of diseased people acutely by up to 30% of starting values.

CONCLUSION

A large series of studies indicate undoubtedly that smoke inhalation, either active (such as smoking cigarettes) or passive (such as breathing indoor smoke), can cause severe lesions to the cardiovascular system. There is a direct correlation between the level of exposure and cardiovascular morbidity. However, several other variables such as type of smoking, ambient atmosphere and subject's health, may influence cardiovascular function as well as interfere with the results of the experimental procedures. Cigar and pipe smoking are not without risk but are significantly less hazardous than cigarette smoking. Habitual heavy use of cigarettes is a major risk factor for coronary atherosclerosis, aortic aneurysms and peripheral arterial disease. Our studies also showed more advanced alterations of the heart of smokers than those of nonsmokers with the same disease.While we can argue about the amount of damage caused by cigarette smoking on cardiovascular system, we cannot deny the evidence that it occurs. No wonder cigarette smoking has been declared as "the single most important environmental factor contributing to premature mortality in the United States"[8].

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