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Effect of Smoking on Development of Coronary Heart Disease

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Abstract: *Cigarette smoking is a major and the single most modifiable risk factor for coronary heart disease. Cigarette smoking increases risk 2 to 3 fold and interacts with other risk factors to multiply risk. The overall examination of the incidence, prevalance, mortality, natural history and risk factors of coronary heart disease suggests the greatest benefits will be from a preventive approach. Present study is done to assess the risk of coronary heart disease in people who are smoking along with other risk factors like obesity , hypertension and hyperlipidemia.*

Materials And Methods: *men who were normotensive and non-smokers with normal cholesterol levels as controls(20) and cases of hypercholesterolemia and non smokers as 1 group(20) and smokers group 2(20). Total cholesterol , HDL, systolic blood pressure, fasting blood pressure are noted.*

Results: *Risk of landing in metabolic syndrome is assessed based on NCEP-ATP-III risk assessment chart . In this study of controls are at low 10 year risk of developing coronary heart disease at average of 2.80 with S.D. ± 1.16. The cases of Group-I are at intermediate risk at average of 11.36, S. D. ± 2.18. Cases of Group-II(smoking) are at high risk at average of 23.05, S. D. ± 2.70.*

Conclusion: *So, our study shows that there will be increase in risk of development of heart diesase with smoking and other risk facors . Assessment of the risk of heart disease helps in prevention of heart disease, so the management of modifiable risk factors by taking necessary preventive measures and life style modifications can help in decreasing the mortality and morbity of heart disease in the early stages itself.*

Keywords: *metabolic syndrome, NCEP-ATP-III risk assessment chart, smoking, coronary heart disease, hyperlipidemia.*

I. Introduction

Coronary heart disease is the most important cause of heart disease and the single most important cause of death in many countries. By 2020 it is estimated that it will become the major cause of death in all regions of the world. Disease of the coronary arteries is almost always due to atheroma and its complications, particularly thrombosis (1). The life time risk of developing CHD after age 40 is 49% in men and 32% in women (2).

Metabolic syndrome is a cluster of conditions which includes increased blood pressure, a high blood sugar level, excess body fat around waist and abnormal cholesterol levels that occur together, increasing your risk of heart disease, stroke and diabetes. Cigarette smoking is patho genetically a cholesteral-dependent risk factor and acts synergistically with other risk factors substantially increasing the risk of coronary heart disease (3,4). A dose related and potentially reversible impairment of endothelium-dependent vasodilation was found in health young adults who smoked cigarettes. Smoking also contributes to coronary artery spasm(5,6). Cigarette smoking increases risk 2 to 3 fold and interacts with other risk factors to multiply risk. The overall examination of the incidence, prevalance, mortality, natural history and risk factors of metabolic syndrome suggests the greatest benefits will be from a preventive approach. A multifactorial approach to risk reduction offers the best opportunity for saving patients at high risk and preventing the development of high risk status in the first place(7). The key parameter for risk assessment for medical intervention is the absolute risk that in the probability of developing metabolic syndrome over a finite period. Some important fixed risk factors of metabolic syndrome are age, male sex and family history and other modifiable risk factors are smoking, hypertension, lipid disorders, diabetes mellitus, haemostatic variables, sedentary life style, obesity and atherogenic diet(1). Framingham scores are used to estimate the absolute risk for the development of CHD over next decade (8). Present study is done to assess the risk of heart disease in people who are smoking along with other risk factors like obesity , hypertension and hyperlipidemia.

Serum thiocyanate - a marker of exposure to smoke, measured postpartum in PDAY study, when evaluated microscopically, established plaques appear to be more rapidly progressing and thus reaching an advanced stage of the disease earlier incase of smokers. A coronary thrombus is more frequently found in smokers than in non-smokers dying suddenly of CHD(9). Strong dose-responsive relationships between cigarette smoking and CHD have been observed in all racial groups. Cigarette smoking increases risk 2 to 3 fold and interacts with other risk factors to multiply risk. There is no evidence that filters or other modifications of the cigarette reduce risk(10). Passive smoking appears to be associated with a small increase in the risk of

CHD. In a recent meta-analysis of 18 epidemiologic studies, exposure to tobacco smoke by non-smokers was consistently associated with a 20% to 30% increase in risk(11)..CO is a colorless, odorless gas produced during incomplete combustion of tobacco. It has 200 times higher affinity for Hb than oxygen does and it impairs release of oxygen from hemoglobin to peripheral tissues. CO also binds to other heme-containing proteins such as myoglobin and cytochrome oxidase. Nicotine is an important constituent of cigarette smoke. It is an alkaloid that readily crosses the blood-brain barrier and stimulates nicotine receptors in the brain and in responsible for tobacco addiction.

It is also responsible for the acute pharmacological effects associated with tobacco use which are most likely mediated by catecholamines. These effects are:

1. Increased heart rate and blood pressure.
2. Increased coronary artery blood flow.
3. Increased contractility and cardiac output.
4. Mobilization of free fatty acids.

Smoking contributes to cardiac arrest by

a. Increasing platelet adhesion and aggregation.
b. Triggering arrhythmia and By causing imbalance between the demand of oxygen and supply to the myocardium(12).

II. Materials And Methods

Controls - men were selected as those who are not hypertensive, not smokers and whose cholesterol levels are in normal range. For the present study all men Patients were selected who are not hypertensive and not smokers are taken as group-I, Smokers as group-II.

Total 60 cases are taken of which, Controls - 20 cases ,Hypercholesterolemia - 20 cases(Group-I) and Smokers - 20 cases(Group-II). All patients and Controls who were men for present study are asked to come to Biochemistry Laboratory on overnight fasting at 9 AM. After brief clinical examination, ie. taking relevant history, recording BP and anthropometric measurements - height, weight, waist cercumferace - blood sample was taken for measurement of fasting glucose and for lipid parameters.

III. Results

In the present study assessment of risk of coronary heart disease for men was done. Assessment of CHD risk is done for all cases according to NCEP - ATP III guide lines with Framingham scoring points for risk factors.

Table1: Controls

S. No.	Age	Total Cholesterol	HDL	S.B.P.	Smoking	FBS	Risk (%)
1	50	180	42	110	-	70	3
2	44	162	44	112	-	68	1
3	46	158	45	110	-	66	1
4	52	148	48	108	-	60	2
5	54	170	50	110	-	65	4
6	48	168	46	112	-	62	2
7	50	150	50	110	-	70	2
8	46	170	45	112	-	72	2
9	56	182	52	110	-	67	5
10	54	160	50	114	-	68	4
11	52	158	48	110	-	72	3
12	48	160	45	108	-	68	2
13	50	170	48	110	-	65	3
14	52	152	50	108	-	60	3
15	46	172	46	110	-	68	2
16	49	168	52	106	-	70	2
17	52	174	48	110	-	66	3
18	50	170	44	110	-	70	3
19	54	164	50	112	-	72	4
20	56	170	46	110	-	68	5

Average	165.05	47.74	110.10	-	67.35	2.80
SD+	9.44	2.60	1.82	-	3.60	1.16

Table2: Hyperlipidemia

S. No.	Age	Total Cholesterol	HDL	S.B.P.	Smoking	FBS	Risk (%)
1	54	260	32	122	-	88	12
2	50	272	34	120	-	85	10
3	48	280	30	118	-	90	10
4	46	254	32	120	-	86	7
5	56	248	30	118	-	88	13
6	58	270	28	110	-	86	16
7	50	265	32	116	-	84	10
8	46	270	30	118	-	90	8
9	54	258	31	110	-	86	11
10	52	240	32	114	-	85	9
11	50	256	30	116	-	82	10
12	53	272	30	110	-	84	12
13	50	260	28	114	-	85	10
14	54	250	32	118	-	80	11
15	57	235	31	120	-	88	13
16	58	248	30	110	-	82	13
17	52	272	33	120	-	87	12
18	54	258	32	118	-	90	12
19	56	270	30	114	-	82	14
20	58	264	31	110	-	84	14
Average		**260.10**	**30.90**	**115.80**	**-**	**85.60**	**11.36**
SD+		**11.80**	**1.52**	**3.90**	**-**	**2.87**	**2.18**

Table3: Smokers

S. No.	Age	Total Cholesterol	HDL	S.B.P.	Smoking	FBS	Risk (%)
1	42	242	26	126	+	96	21
2	44	234	24	128	+	98	22
3	50	226	27	120	+	94	21
4	59	213	25	130	+	97	26
5	46	212	28	124	+	92	17
6	55	218	26	130	+	95	25
7	50	220	29	120	+	90	19
8	58	230	25	124	+	88	26
9	48	222	26	128	+	93	22
10	45	254	25	120	+	96	25
11	50	220	24	124	+	92	23
12	58	235	26	128	+	95	27
13	53	238	24	120	+	90	26
14	52	224	26	12	+	92	23
15	50	211	27	130	+	94	20
16	52	220	25	120	+	96	22
17	56	232	29	124	+	88	23
18	45	240	24	120	+	95	23
19	58	230	25	126	+	97	27
20	56	225	28	124	+	92	23
Average		227.30	25.95	124.40	+	93.50	23.05
SD+		11.10	1.60	3.70	+	2.94	2.70

Values of significance:

Table4: control verses group 1 (hyperlipidemia)

Total cholesterol	HDL	SBP	FBS
P<0.001	0.001	0.01	0.001

Table5: Control verses group 11(smokers)

Total cholesterol	HDL	SBP	FBS

P < 0.001	0.001	0.001	0.001

Integration of risk:

Patients without manifestations of CHD who have a 10 year risk for MI of greater than 20% are at high risk.

Intermediate risk patients have a 10 year risk for MI of 10% to 20%.

Low risk patients are those whose 10 year risk for MI is less than 10%.

In this study of controls are at low 10 year risk of developing MI at average of 2.80 with S.D. ± 1.16. The cases of Group-I are at intermediate risk at average of 11.36, S. D. ± 2.18. Cases of Group-II(smoking) are at high risk at average of 23.05, S. D. ± 2.70. Risk assessment of coronary heart disease based on NCEP ATP - III risk assessment chart states that with smoking and other increasing risk factors there is increase in the risk of development of heart disease in the next 10 years.

IV. Discussion

Gerald M. Reaven has hypothesized that insulin resistance impairs glucose uptake and compensatory hyperinsulinemia are the common metabolic basis for a cluster of coronary risk factors, particularly smoking, hypertension, diabetes, hypertriglyceridemia, low HDL predominance of small dense LDL and a prothrombotic state with elevated levels of plasma fibrinogen, plasminogen activator inhibitor 1 (PAI-1) and factor VII (12).

Cessation of smoking is associated with a precipitous fall in CHD events. In a previous smoker, the relative risk declines nearly to that of a non - smoker in a year or less (13). It is estimated that a 35 year old who quits smoking extends survival by 3 to 5 years, with much of the improved life expectancy caused by a reduction in CHD deaths (14). Interventions to achieve smoking cessation are among the most cost - effective in either primary or secondary prevention with or without the use of nicotine replacement therapy. The home and work environment to which patients return should be smoke free, both to encourage cessation and to reduce the risk from passive smoking. Smoking cessation clinical practice guidelines (15) emphasize that tobacco use status be documented in every patient and that every smoker should be offered one (or) more of three effective treatment interventions. Even a brief intervention may be effective and should at a minimum, be provided to every patient who uses tobacco. Three elements of a treatment program found to be effective include :

R Social support
R Skills training / problem solving and
R Nicotine replacement.

Addiction to tobacco is a major barrier to cessation, and a number of pharmacologic agents can be recommended as an adjunct to a concurrent behavioral intervention on the basis of clinical traits demonstrating significantly increased rates of smoking cessation (16). Nicotine gum, nicotine inhaler, nicotine nasal spray and nicotine patch are all first - line drugs to prevent nicotine withdrawal.

A huge reduction in risk resulting from smoking cessation in the cardio vascular disease patient provides a strong rationale for sustained and intense efforts to be expended.

Strategies for successful cessation of cigarette smoking. The four A's by fiore M et. al. and Pearson TA (15,17).

Ask - Systematically identify all tobacco users at every visit. Identify patients with nicotine addiction.

Advise - Provide a clear, strong and personalized message, urging every tobacco user to quit. Review benefits of quitting and risk of continuing Assess patients Willingness to quit.

Assist - Have the patient develop a quit plan, including setting a quit date, identifying sources of support for cessation for family and friends, removing tobacco and other cues from the home and work environment. Provide counseling, information materials and other behavioral interventions recommend use of pharmacology to prevent nicotine withdrawal.

Arrange - Provide a remainder on the quit date, see the patient shortly after the quit date to assess success. If unsuccessful, identify barriers and solutions to their removal.

Dietary interventions should be the initial step in the treatment of dyslipidemia, hypertension, diabetes and obesity. For a patient with hyperlipidemia, less than 7% of calories from saturated fat and less than 200 mg of dietary cholesterol per day are suggested. Mono unsaturated fats and omega-3 fatty acids from fish may be a beneficial source of calories, as compared with carbohydrate (18). The current dietary recommendations emphasize a well balanced diet low in saturated fat, cholesterol and sodium which are rich in fruits and vegetables (86).

The Lyon Diet Heart Study, with Mediterranean - type diet enriched in - linolenic acid demonstrated a 65% reduction in recurrent cardiac events and death over a 4 years period of follow-up (87). Two older clinical trials of long - term inpatients demonstrated reductions in coronary end points of 34 to 50 percent among patients on low saturated fat and cholesterol diets (88,89).

V. Conclusion

In the present study parameters of cases of smokers, hypertensives and hyperlipidemia are taken along with control and their risk is assessed. Smokers had high risk (Average of 23.05 with S. D. ± 2.70) when compared to other groups of cases which showed increased levels of total cholesterol and decreased HDL - cholesterol levels and levels of SBP and FBS are in high normal range. Passive smoking may also contribute to this considerable increase in risk.. Hypercholesterolemia cases show increased levels of total cholesterol, decreased HDL - cholesterol and normal range of SBP & FBS. The risk of development of coronary heart disease in future is moderate of Average of 11.35 with S. D. ± 2.18. Controls showed a little risk of development of heart disease of Average of 2.80 with S. D. ± 1.16 which may be due to their age and male sex.

So, our study shows that there will be increase in risk of development of heart disease with increase in number of risk factors. Assessment of the risk of heart disease helps in prevention of heart disease, so the management of modifiable risk factors by taking necessary preventive measures and life style modifications can help in decreasing the mortality and morbity of heart disease in the early stages itself.

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