ATHEROSCLEROTIC LESIONS IN CIGARETTE SMOKERS AND HUKKA SMOKERS: A COMPARATIVE HUMAN AUTOPSY STUDY

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ABSTRACT

Objective: *To assess the different morphological types of atherosclerotic lesions in cigarette smokers and Hukka smokers in our population.*

Methodology: *This descriptive study was conducted at the mortuary of King Edward Medical University Lahore and Department of Pathology Allama Iqbal Medical College Lahore from June 2007 to July 2009. Heart, aorta coronary arteries and renal arteries were collected from dead bodies. One to four areas of tissue were taken from aorta and each artery in all cases. Sections were prepared from paraffin blocks. These were stained with Haematoxylin and Eosin stain. Special stains were also performed to differentiate all the components of atherosclerotic lesions.*

Results: *A total of 130 human autopsies were carried out in this study. Out of these, 84 were smokers (62 cigarette and 22 hukka somkers). The analysis was carried out on these 84 cases. The mean age of smokers was 48.63±16.079. Fibrolipid plaques and complicated/ calcified lesions were seen in predominantly more cases in cigarette smokers, than hukka smokers in aorta, coronary arteries and renal arteries.*

Conclusion: *This study shows distribution of different atherosclerotic lesions in cigarette smokers and hukka smokers in our population. The raised atherosclerotic involvement in cigarette smokers as compared to hukka smokers is evident from the results.*

Key wards: *Atherosclerosis, Cigarette, Hukka, Smokers, Arteries.*

INTRODUCTION

The exact role of cigarette smoking as a risk factor in atherosclerosis has been controversial[1]. Cigarette smoking as a risk factor in coronary atherosclerosis provides the fact that there is a direct relationship between the severity of the disease process and the number of cigarettes smoked per day. The platelet stickiness and aggregation increases after cigarette smoking[1]. On the contrary, no elevation in lipid levels was observed in young adult male smokers by Konttinen[2]. However, currently it is believed that cigarette smoking does increase the incidence of atherosclerosis[3]. Stopping pregnant women from smoking may help to prevent coronary heart disease in their children in later life[4]. Carbon monoxide from cigarette by causing anoxic damage to the arterial lining, accelerates atherogenesis in the presence of hyperlipidemia[5].

METHODOLOGY

This descriptive study was conducted at the mortuary of King Edward Medical University Lahore and Department of Pathology Allama Iqbal Medical College Lahore from June 2007 to July 2009. A total of one hundred and thirty human autopsies were carried out during this study.

Selection of Dead Bodies: All the dead bodies included in this study were examined in the interval which ranged from 4-10 hours between the death and autopsy. Dead bodies of men, women and children were included at random i.e., on the basis of availability. In each case, the relevant history was obtained from the close relatives of the deceased. Autopsies were performed. Heart, aorta, coronary arteries and renal arteries were collected and included in this study. Performa for relevant history and autopsy findings included name,

gender, date of birth, place of residence, occupation, any medical are before death, date of death, mode of death (Accidental/Non-accidental), clinical diagnosis made before death and smoking habits (Cigarette/Hukka/Others).

Grading of Atheroma: Gross sections of coronary arteries were graded by one of the four scores according to the degree of atheromatous narrowing, Grade-I, upto 25% narrowing, Grade-II, 26-50% narrowing, Grade-III, 51-75% narrowing and Grade-IV greater than 75% narrowing. Complete occlusion with haemorrhage, ulceration, thrombosis and calcification were recorded separately. In addition, major degree of narrowing in each branch was noted; isolated areas of narrowing were specified as "Focal" and distance from origin of artery was noted. In all the 130 autopsies aorta, coronary arteries and renal arteries were examined. 1-4 sections were taken from aorta for histological examination from the Arch of aorta; Above the celiac artery level (thoracic); At renal arteries level (abdominal); and Below renal arteries level (abdominal).

In addition, 1-4 section from each of the coronary arteries and renal arteries were taken. For histological examination tissue processing was done. On an average, 7-8 slides were prepared from each block by taking ribbons of tissue. Paraffin section were stained using Haematoxylin and Eosin stain, von kossa's staining technique, periodic acid Schiff (PAS) reaction, Toludine blue stain and Peral's Prussian blue stain.

Smoking habits related to cigarettes, hukka or others (pipe, cigars) were found out by interviewing the living relatives of the deceased persons (Verbal autopsy method). Habitual smokers were included but the exact duration and frequency of the smoking habits were not exactly known to the closest relatives of the deceased. Amongst smokers only cigarette and hukka smokers were found in this study. Different categories of atherosclerotic lesions such as fatty streaks, fibrolipid plaques, complicated (ulceration, haemorrhage and thrombosis) and calcified lesions were noted. Fibrolipid plaques, complicated and calcified lesions were named as the raised lesions. Atherosclerotic ulcers were seen with ragged edges. No case of aneurismal dilatation or rupture of aorta was observed during the study.

RESULTS

In a total of 130 cases where aorta, coronary arteries and renal arteries were collected, ninety were males and forty females. A total of 84 were found to be smokers; 62 cases (73.8%) showed the history of cigarette smoking and 22 (26.2%) were hukka smokers. The analysis was carried out on these 84 cases. The mean age of smokers was 48.63$\pm$16.079. There were 58 males and 4 female cigarette smokers while there were 15 male and 7 female hukka smokers (Figure 1). Fatty streaks were more common in cigarette smokers than in hukka smokers in aorta, coronary arteries and renal arteries. Fibrolipid plaques were seen in more cases in cigarette smokers, than hukka smokers in aorta, coronary arteries and renal arteries (Figure 2, 3). Complicated and calcified lesions were present in more number of cases in cigarette smokers than in hukka smokers in aorta, coronary arteries and renal arteries. (Table 1).

DISCUSSION

In this study, fatty streaks were present in

Figure 1: Gender distribution of the cigarette and hukka smokers

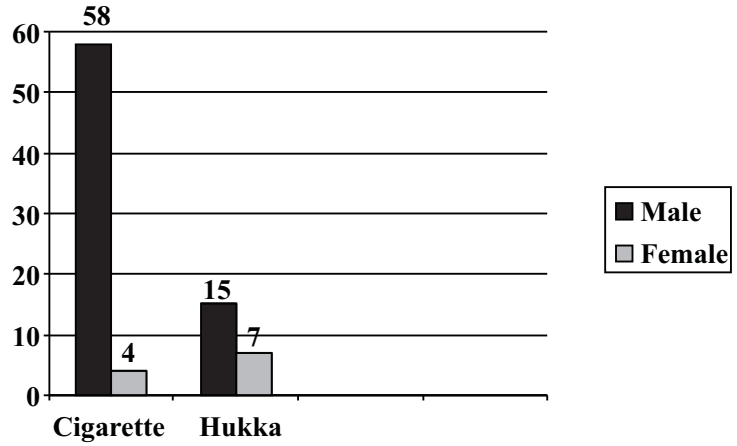


Table 1: Number And Percentage Distribution of Atherosclerotic Lesions in Aorta, Coronary Arteries and Renal Arteries InRelation To History of Smoking Habits

Blood Vessels	Fatty streaks				Fibrolipid plaques				Complicated lesions				Calcified Lesions			
	Cigarette Smokers		Hukka Smokers		Cigarette Smokers		Hukka Smokers		Cigarette Smokers		Hukka Smokers		Cigarette Smokers		Hukka Smokers	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Thoracic Aorta	46	74.2	6	27.3	25	40.3	4	18.2	10	16.1	2	9.1	7	11.3	1	4.5
Abdominal Aorta	35	56.5	6	27.3	49	79.0	7	31.8	31	50.0	4	18.2	15	24.2	2	9.1
Anterior Descending Left Coronary Artery	13	21.0	3	13.6	41	66.1	6	27.3	8	13.0	2	9.1	4	6.5	-	-
Circumflex Left Coronary Artery	9	14.5	2	9.1	35	56.5	5	22.7	4	6.5	1	4.5	1	1.6	-	-
Right Coronary Artery	2	3.2	-	-	3	4.8	1	4.5	1	1.6	-	-	-	-	-	-
Right Renal Artery	20	32.3	3	13.6	18	29.0	2	9.1	7	11.3	-	-	4	6.5	-	-
Left Renal Artery	20	32.3	3	13.6	12	19.4	1	4.5	6	9.7	-	-	4	6.5	-	-
Right Common Iliac artery	3	23.1	1	16.7	10	77.0	2	33.3	7	53.8	1	16.7	5	38.5	1	16.7
Left common Iliac artery	3	23.1	1	16.7	10	77.0	2	33.3	6	46.2	1	16.7	5	38.5	1	16.7
Mean Incidence in all vessels		**31.1**		**15.3**		**49.9**		**20.5**		**23.1**		**8.2**		**14.8**		**5.2**

Figure 2: Atherosclerotic lesion in aorta showing cholesterol crystal clefts with free lipid pool. Haemotoxylin and Eosin stain x 200

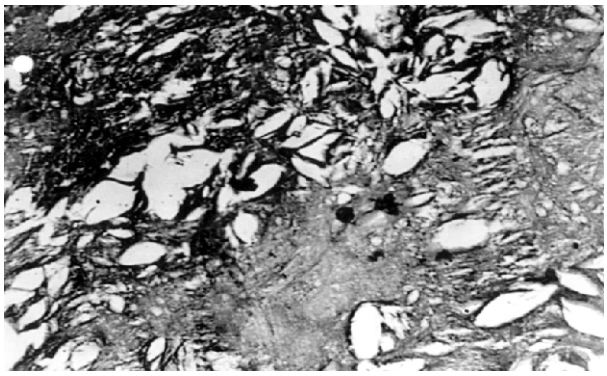
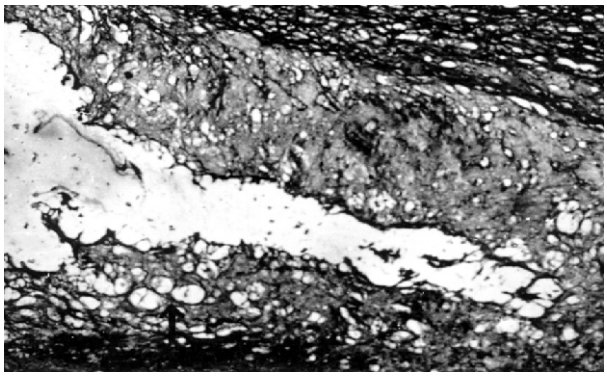


Figure 3: Atherosclerotic lesion in coronary artery showing lipid pool and foam cells. Haemotoxylin and Eosin stain x 300.



predominantly more cases in cigarette smokers than hukka smokers in aorta, coronary arteries and renal arteries. Fibrolipid plaques, complicated and calcified lesions were present in more number of cases in cigarette smokers than in hukka smokers in aorta, coronary arteries and renal arteries. Independent association between cigarette smoking and aortic and coronary atherosclerosis has been established[6, 7]. Supporting these observation[8, 9] also described that cigarette smokers have shown about a two-fold excess incidence of coronary heart disease than non-smokers. Carbon monoxide because of its affinity for haemoglobin and myoglobin, interferes with the transport and tissue utilization of oxygen. This causes anoxic damage to the vessel wall and in the presence of other factors such as hyperlipidemia accelerates atherosclerosis. Catecholamine's are released by the nicotine that increases the blood pressure[5] also proposed the same view and described that catecholamine release promote the thrombotic tendencies[10]. They explained that nicotine actively raised systolic blood pressure, heart rate and cardiac output[11,12]. Nicotine stimulated catech-

olamine release may increase platelet stickiness and aggregation. Carboxy haemoglobin increases vessel wall hypoxia and permeability such increase permeability of vessel wall to lipoprotein may promote atherosclerosis. Carboxy haemoglobin also increase the tendency of occurrence of thrombosis by increasing platelet adhesiveness and by stimulating erythropoietin which increases blood viscosity.

Nicotine also stimulates the sympathetic system and consequently leads to the mobilization of fatty acids, cholesterol and other lipids from fat depots, with the elevation of their level in the blood. It has also been described that tobacco component other than nicotine and carbon monoxide causes a small decrease in exercise performance in angina pectoris[13]. It is further explained that pipe smokers have a relatively high nicotine intake but little if any risk of death from coronary heart disease due to different inhaling patterns, strongly suggests that nicotine is not the major cause of the excess risk found in cigarette smokers. Nicotine is more toxic when it is absorbed through the pulmonary alveoli than when absorbed through the buckle mucosa as in pipe smokers. It has also been observed that Carboxy haemoglobin levels in pipe smokers fall between those in smokers and non-smokers, explains but COHB is responsible for the excuse of coronary heart disease in cigarette smokers[14,15]. It is suspected that some other constituent of cigarette smoke that is correlated with CO may be the principle etiological agent. On the other hand described that stopping cigarette smoking does not reduce the risk of coronary heart disease[16]. It is also mentioned that serum total cholesterol, Low-density lipoprotein – cholesterol and high-density lipoprotein cholesterol fractions showed no differences relating to smoking habits[2].

Atherosclerotic disease is associated with risk of aortic aneurysm in the general population. In addition, cigarette smoking appears to have a direct effect on the risk of aortic aneurysm which is independent of atherosclerosis[17]. In smokers the bulk of events are the result of a progressive disease of the arterial wall known as atherosclerosis and the acute repute of an atherosclerotic lesion. Atherosclerosis is the accumulation of fats, oxidized cholesterol and other material in the portion of the arterial wall known as the intima. This accumulation occurs over decades. A heart attack or thrombotic stroke occurs when an atherosclerotic lesion ruptures and a blood clot (thrombus) is formed that suddenly restricts the flow of blood to the heart of brain[18, 19]. One of the most important modifiable risk factors is smoking. (using other forms of tobacco, such as snuff and chewing tobacco, also increase risk). A smoker's risk of developing coronary artery disease is directly related to the amount of tobacco smoked. The risk of a heart attack is increased threefold in men and six fold in women who smoked 20 or more cigarettes per day compared with nonsmokers. In people who already have a high risk of heart disease, tobacco use in particularly dangerous. Tobacco use decreases the level of high-density lipoprotein (HDL) cholesterol- the good cholesterol and increase the level of low density lipoprotein (LDL) cholesterol the bad cholesterol. Smoking increases the level of carbon monoxide in the blood, which may increase the risk of injury to the lining of the artery's wall. Tobacco use causes arteries already narrowed by atherosclerosis to constrict, further decreasing the amount of blood reaching the tissues. In addition, tobacco use increases the blood's tendency to clot, so that it is increases the risk of peripheral arterial disease (atherosclerosis affecting arteries other than those that supply the heart and brain) [20, 21]. It is concluded that healthy adolescents frequently exposed to tobacco smoke have arterial changes associated with preclinical atherosclerosis and increased apo lipoprotein B (apo B) levels [22].

The mode of smoking in hukka smokers and in pipe is almost similar with the exception that in hukka most of the injurious contents of the smoke such as nicotine etc., are trapped in the water container. Therefore, in hukka smokers although the absorption is through buckle mucosa as in pipe smokers, the contents of the smoke are far less concentrated in nicotine and carbomonoxide etc. this facts helps in explaining the significantly mild atherosclerotic lesions amongst hukka smokers then cigarette smokers in whom the absorption is through bronchial tree.

CONCLUSION

This study shows distribution of different atherosclerotic lesions in cigarette smokers and hukka smokers in our population. The raised atherosclerotic involvement in cigarette smokers as compared to hukka smokers is evident from the results.

REFERENCES

1. Woolf N. Atherosclerosis. In: Pomerance A, Davies MJ, editors. The Pathology of the heart. Edinburgh: Blackwell Scientific Publications; 1975. p. 127-66.
2. Konttien A. Cigarette smoking and serum lipide in young men. Br Med J 1962;1:1115-6.
3. Reid DD, Hamilton PJS, McCartney P, Rose G, Jarrett RJ, Keen H. Smoking and other risk factors for coronary heart disease in British Civil Servants. Lancet 1976;2: 979-84.

4. Asmussen I. Arterial changes in infants of smoking mothers. Postgrad Med J 1978;54:200-5.

5. Kannel WB, Doyle JT Fredrickson DT, Harlan WR Jr. Report of the adhoc committee on cigarette smoking and cardiovascular disease for health professionals. Circulation 1978; 57:404-5.

6. Rhoads GG, Blackwelder WC, Stemmerman GN, Hayashi T, Kagan A. Coronary risk factors and autopsy findings in Japanese – American men. Lab Invest 1978;38:304-11.

7. Vlietstra RE, Frye RL, Kronmal RA, Sim DA, Tristani FE, Killip T. Risk factors and angiographic coronary artery disease report from the coronary artery surgery study(CASS). Circulation 1980;62:254-61.

8. Friedman GD, Petitti DB, Bawol RD, Siegelaub AB. Mortality in cigarette smokers and quitters. Effect of base-line differences. New Eng J Med 1981;304:1407-10.

9. Kannal WB. Cigarettes, coronary occlusions and mycoardial infarction. JAMA 1981;246 :871-2.

10. Friedman GD. Cigarette smoking and coronary heart disease, new evidence and old reactions. Am Heart J 1980;99:398-9.

11. Castelli WO, Garrison RJ, Dawber TR, McNamara PM, Feinleib M, Kannel WB. The filter cigarette and coronary heart disease: the Framingham study. Lancet 1981:2:109-13.

12. Hartz AJ, Barboriak PN, Anderson AJ, Hoffmannn RG, Barboriak JJ. Smoking, coronary artery occlusion and nonfatal myocardial infarction. JAMA 1981;246:851-3.

13. Aronow WS. Effect of non-nicotine cigarettes and carbon Monoxide on angina. Circulation 1980;61:262-5.

14. Wald NJ, Idle M, Boreham J, Bailey A. Serum continine levels in pipe smokers; heart disease. Lancet 1981;2:775-7.

15. McCusker K, McNabb E, Bone R. Plasma nicotine levels in pipe smokers. JAMA 1982;248:577-8.

16. Seltzer CC. Smoking and coronary heart disease: what are we to believe? Am Heart J 1980;100:275-9.

17. Lee AJ, Fowkes FG, Carson MN, Leng GC, Allan PL. Smoking stherosclerosis and risk of abdominal aortic aneurysm. Eur Heart J 1997;18:671-6.

18. Dwyer JH, Navab M, Dwyer JM, Hassan K, Sun P, Shircore A. Oxygenated carotenoid lutein and progression of early atherosclerosis: the Los Angeles atherosclerosis study. Circulation 2001;103:2922-7.

19. Rifkind BM, Grouse LD. Cholesterol redux. JAMA 1990;264:3060-1.

20. Jules YTL. Atherosclerosis heart and blood disorders: Merck Manual home edition. New Jersey: Whitehouse Station; 2008.

21. Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K, et al. Risk factors for early myocardial infarction in south asians compared with individuals in other countries. JAMA 2007;297:286-94.

22. Kallio K, Jokinen E, Saarinen M, Hamalainen M, Volanen I, Kaitosaari T, et al. Arterial intima-media thickness, endothelial function, and apolipoproteins in adolescents frequency exposed to tobacco smoke.Circ Cardivoasc Qual Outcomes 2010;3:196-203.

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