BLOOD CHOLESTEROL AND HDL CHOLESTEROL IN CIGARETTE SMOKERS

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Abstract : 24 cigarette smokers were investigated for their blood cholesterol and HDL cholesterol. They had elevated total cholesterol compared to age and sex matched controls. 21 smokers out of 24 had significant decrease of HDL cholesterol. It is suggested that smoking, which is a major risk factor for coronary heart disease, might act through its effect on total cholesterol and HDL cholesterol.

Key words : cholesterol lipoproteins smoking atherosclerosis coronary artery disease

INTRODUCTION

Cigarette smoking is considered a major risk factor for coronary heart disease (1). It has also been shown that a decrease of high density lipoprotein (HDL) cholesterol is a risk factor for the same (2). In normal individuals, HDL cholesterol should be about 20 per cent of the total cholesterol (3). Craig et al (4) have analysed data from 54 studies on blood lipid changes in smokers. These studies are mostly on Western subjects. We have studied serum lipids in Indian smokers to investigate any relationship between smoking and serum cholesterol and the HDL fraction.

Cholesterol levels in blood are influenced by diet. Hence changes in dietary pattern might affect chelesterol level in addition to cigarette smoking. As the normal diet of Western population has much larger amounts of fat than that of Indians, it was of interest to study the effect of smoking on Indian subjects consuming 10 to 15% of fat in their diet. HDL cholesterol levels may also be affected by physical exercise, which may not be the same in Indian and Western populations.

Hence, though there have been many studies, we were interested in finding whether the reported

conclusions apply to Indians. Apart from the deranged cholesterol metabolism, other hazards like Cadmium (5), Carbon monoxide and Nicotine toxicity and depletion of Vitamin C (6) have been noticed in cigarette smokers.

METHODS

The study waas conducted on 24 male smokers (height 157 cm-175 cm, weight 55-69 kg). They were all vegetarians and belonged to the age group of 18 to 32 years. They were free from diseases like diabetes mellitus, myxodema, atherosclerosis and did not consume alcohol. They were also indulging in equivalent physical exercise. Their diet consisted of 70% carbohydrate as rice, wheat, potatoes etc, 18% protein as pulses, dahl and milk, and 12% fat as butter, ghee and edible oils. They did not consume refined oils. The Non-smoker control volunteers were also 24 in number, and were age and sex matched, and had comparable diet and physical activity.

Blood was taken at the same time in all cases and the investigations were done within 2 hours of collecting blood. Total cholesterol was estimated by the method of Schoenheimer and Sperry modified by Venugopala Rao and Ramakrishnan (7), while

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HDL cholesterol was analysed by the technique of polyanion precipitation with Heparin and managanous chloride and estimated as cholesterol in the supernatant. This measures only HDL cholesterol (8).

RESULTS AND DISCUSSION

Cholesterol in blood has been reported to increase in smokers to the extent of 4.5% by the previous workers. But in our studies it was found to be higher by 18.1% as compared to controls. This increase has been observed even when the dietary fat is only 12%, which is only about one fourth of the fat consumed daily by the Westerners. Hence it has been confirmed that increase of cholesterol is only due to cigarette smoking and independent of fat content of the diet. This conclusion has been arrived at by previous workers in animal experiments and withdrawal studies. The marked increase of serum cholesterol in Indian smokers in spite of a relatively low dietary fat intake may be due to their smoking cigarettes without filters. More of nicotine is likely to be inhaled if filters are not used.

HDL cholesterol of serum has been shown to decrease by 8.9% in smokers by previous workers while in the present work it was of the order of 32.3%. An increase of cholesterol (as LDL) might be suggestive of an increase of VLDL from which LDL is formed by delipidation. A greater increase of LDL and VLDL may cause a greater decrease of HDL as there is a reciprocal relation between the concentration of VLDL and HDL.

The interesting finding is therefore a greater increase of total cholesterol and greater decrease of

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Category	Total cholesterol (TC)	HDL cholesterol	$\frac{HDL}{TC}$ %
Normal	171.5±4.8	43.6±2.3	25.9±1.5
Cigarette- smoking	202.6±8.8*	29.5±1.1**	15.3±0.9**

Values are mean of 24 males \pm S.E. *P<0.001; **P<0.01.

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HDL cholesterol in Indian smokers compared to Westerners. As both an increase of total cholesterol and a decrease of HDL cholesterol are risk factors for coronary heart disease, Indian smokers appear more susceptible to heart disease as compared to their Western counterparts.

The mechanism of increase of blood cholesterol is through an increase of Free fatty acids of blood. In smokers, the nicotine ingested stimulates the secretion of catecholamines. These hormones increase the FFA by lipolysis of adipose tissue fat. The FFA reaching the liver are esterified as Triacylglycerol and cholesteryl esters which are secreted into the blood stream as VLDL which gets converted to LDL in circulation. The greater the release of FFA, the greater the levels of LDL and cholesterol.

The decrease of HDL may be due to 3 factors. Increase of VLDL will cause a decrease of HDL due to poorer availability of phospholipid remnants from VLDL for HDL formation. It may also be due to decreased Apo A content, as Apo A is needed for the formation of HDL, and finally, diminished lecithin cholesterol acyl transferase (LCAT) needed for the formation of HDL (9). A decrease of Apo A can cause a diminished activity of LCAT as Apo A is its activator (10). Again, an accumulation of Cadmium may cause a decrease of both Apo A and LCAT (which are proteins) by heavy metal precipitation.

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