Original Article

Study of apolipoproteins in alcoholics

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Abstract

50 alcoholic patients from psychiatry department are selected and 50 non alcoholics as control. Their blood samples are collected. The present study aims to measure the biochemical parameters in alcoholics and to examine how these factors contribute to heart stroke, CAD, atherosclerosis, CHD, hypertension and other complications and to search for new abnormality associated with abnormal lipid profile in alcoholism. It can be concluded that rigid metabolic control of alcoholism (hypertension, hypertriglyceridemia, hypercholesterolemia) will prevent the alteration of lipids and lipoprotein metabolism and also postpone the manifestation of vascular complication with the control of socio complications.

Key Words: Apolipoprotein, alcoholism, phospholipids, lipid profile, atherosclerosis, biomarkers.

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INTRODUCTION

Impact of alcohol on systemic disease and general health is of great concern. Study of biochemical parameters like Apolipoprotein A1, Apo B, lipid profile, Vitamin B6 levels in alcoholics give rise to reliable findings. Water soluble vitamin such as B_1, B_6 have been reported to be deficient in alcoholics. General causes are malnutrition, inadequate nutrients. Particularly lack of water soluble vitamin in the diet, reduced uptake, utilization, increased utilization of nutrient deficiency. Nutrients deficiency therefore virtually inevitable consequences of alcohol abuse, not only alcohol displaces food but also, directly interfere with the body's use of the use of nutrients, make them ineffective even if they are present. Chronic alcoholism exhibits number of the neurological disorders, which are related to the nutritional deficiencies, that are essential for the normal cerebral functioning. Specific vitamin and nutrient deficiencies are arising in chronic alcoholism may resulted into several impairments and tissue damage, mainly neuronal or cardiovascular, in the brain nutritional deficiency in alcoholics also causes neurotransmitter dysfunction and metabolic dysfunction in the brain. Nutritional deficiency in chronic alcoholics frequently leads to mild to moderate longitive impairment, including impairment in perpetual motor skills, visual special functions, learning, memory and absorption and problem solving. Thus attention has to be paid to understand alcoholics from biochemical point of view. The present study aims to measure the above biochemical parameters in alcoholics and to examine how these factors contribute to heart stroke, CAD, atherosclerosis, CHD, hypertension and other complications and to search for new abnormality associated with abnormal lipid profile in alcoholism. This helps us to screen patient properly in their diagnosis and treatment and also give a probable hint towards development of complications

MATERIALS AND METHODS

The subjects of both gender and age groups between 18-70 years are screened for alcoholic patient among the patients admitted in Psychiatry ward. 50 patients are selected for control group who have never consumed alcohol. 50 acute and chronic alcoholics (acute>2 drinks/day with structural and functional changes in liver) (chronic >5-10 years of drinking) are selected for study

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group. Serum is collected and Following parameters are estimated by Wybenga and Pileggis method/Biolab diagnostic kit:
Total cholesterol
HDL
LDL (triglyceride/5 + HDL)
Triglycerides
Phospholipid
VLDL (triglyceride/5)
Apolipoprotein A1 and B by immunotubidiometric method
Vitamin B6
SGOT, SGPT

RESULTS AND DISCUSSION

Table 1: Alcoholic patients with AUD/ALD admitted in psychiatric department JJ Hospital under supervision of Head of Department

Sr. no	Lipids/lipoproteins	Mean± SD	t - value	p- value
1	Cholesterol	171±43.3	28.1	<0.05
2	Triglyceride	105±52.4	14.2	<0.05
3	HDL	44.7±7.4	29.4	<0.05
4	LDL	102.1±40.0	18.1	<0.05
5	VLDL	24.0±21.9	7.8	<0.05
6	SGOT	58.6±46.9	8.8	<0.05
7	SGPT	33.2±23.6	9.9	<0.05
8	VitaminB_6	6.0±5.1	8.3	<0.05
9	Apo A1	121.3±11.1	77.1	<0.05
10	Apo B	113.0±37	21.6	<0.05
11	Apo A1/Apo B ratio	1.15±0.4	19.7	<0.05

There was significant elevation in levels of lipids, triglycerides, cholesterol as compared with non alcoholic control group. The rising tendency of different lipid fraction were noted as the severity of hyperglycemia increases. Hypertriglyceridemia and increased VLDL were the prominent lipoprotein abnormality with commonest being hypertriglyceridemia (type IV). Hypercholesterolemia (type IIb) and combined hyperlipidemia were also observed. Serum phosphoplipids concentration bear a close relationship with the serum cholesterol. Phospholipids also significantly increased in alcoholic patients as compared to controls. Reduction of HDL cholesterol due to replacement of cholesterol moiety by triglyceride and glycosylation of HDL-c leading to increased catabolism. There was no significant difference agewise in serum lipids and lipoproteins in different age groups but increases as the age increases. There was significant difference sexwise in hyperlipidemia. In females it was accentuated. Risk factor for atherosclerosis increases with increase in cholesterol, TG, phospholipid, LDL, VLDL. Obesity adversely affects lipid profile in both alcoholic and non alcoholic groups. Abnormal lipids and lipoproteins favored the occurrence of CAD with female preponderance. However there was no significant correlation between hypertension and lipid profile. Dietary regulation, weight reduction, regular exercises, glycemic control, early management of hypertension would prevent or postpone atherosclerosis related morbidity and mortality events.

CONCLUSION

Thus it can be concluded that rigid metabolic control of alcoholism (hypertension, hypertriglyceridemia, hypercholesterolemia) will prevent the alteration of lipids and lipoprotein metabolism and also postpone the manifestation of vascular complication with the control of socio complications. Our study has some limitations, duration and dosing of alcohol interventions were different, as were the characteristics of the participants. Therefore it is possible that potential confounders such as smoking, physical inactivity, body weight, and diet and other addictions could have affected our findings. Our selection of biomarkers for study was guided by links to cardiovascular pathophysiology. Other biomarkers may be relevance to alcohol's effects on other health conditions- e.g. cancer. Lastly, although we found that alcohol consumption has favorable effects on some biomarkers associated with coronary heart disease, this remains indirect evidence for the mechanisms by which alcohol may cause cardioprotection.

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