ORIGINAL ARTICLE

A COMPARATIVE STUDY OF ATENOLOL AND PROPRANOLOL ON CHEMODYNAMICS AND LIPID PROFILE IN ALCOHOLIC HYPERTENSIVE PATIENTS

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INTRODUCTION: In developing countries particularly in India, the constitutions of the diseases, showing a fatal cause has considerably changed during the recent period. Lifestyle related diseases such as Cancer, Cardio-Vascular, Metabolic and Allergic diseases have accounted for the majority of the diseases showing a fatal course.

The disease structure of Indian People has undergone radical changes in last few years. Tuberculosis which used to be the highest cause of death has decreased greatly. On the other hand incidents of Malignant Neoplasm and Heart diseases have substantially increased. Today these diseases which are closely related to life style of individuals account for 60% of all causes of death. Considering the fact that the natural course of these diseases from onset to death is extensive, it is quite meaningful to reveal the risk factors for the occurrence of Cardio-Vascular diseases.

The mortality rate associated with Cardio-Vascular Heart diseases in Indian People is on the increase from the global aspect. The main risk factors for ischaemic heart disease are hypertension Hyper-cholesterolemia and cigarette smoking. The other factors being obesity alcohol drinking, exercise, stress and Glucose intolerance. As hypertension is now adequately controlled, ischaemic Heart disease can be prevented to a certain degree.

However, the recent National Study of Cardio-Vascular diseases shows higher mean Cholesterol values, therefore, the tend in occurrence of hyper cholesterolemia and ischaemic Heart disease should be watched carefully.

According to the previous studies (Morimotta. K. et al 2000)[1] smokers showed a decrease in HDL by approximately 6% and an increase in LDL as compared to non-smokers. This shows that smoking had adverse influence on lipid balance in blood. On the other hand continuous exercise accompanying weight loss, increased HDL by approximately 5% and decreased LDL by approximately 10%. This shows that exercise is the most effective lifestyle for the lipid balance in blood (Craig. W.Y. et al 1989). Light to moderate alcohol drinking has shown to increase HDL but excess drinking increases TG and thus results in the increase of the risk of cardiovascular disease (Thun M. J. et al 1997).[2]

The other main risk factor of cardiovascular diseases is hypertension. At present the number of hypertension patients is on the increase and approximately 90% of them have essential HT without distinct underlying disease.

The relation between the onset of hypertension to alcohol drinking dietary life, obesity and lack of exercise has been advocated in most literature (Hagberg, J.M. et al 1990).[3] A report on the influence of alcohol drinking on hypertension has shown that diastolic % systolic B.P.

levels were decreased by 3-16mm and 2-8 mm Hg. Respectively by abstinence from drink or by moderation in drinking in HT patients (Potter, J.F. et al 1984).[4]

With regard to influence of dietary life on cardiovascular disease some reports have shown that the dietary sodium over ingestion and Potassium under ingestion have lead to development of H.T. (Fujita T. et al 1980).[5] According to the past studies, reduction in dietary sodium by 100 mmol (5.9gm)/day decreased systolic and diastolic BP levels by 3.7 mm Hg and 0.9mm Hg. respectively. (Midgley, J.P. et al 1996).[6] Some previous reports have shown the influence of potassium administered on hypertension, i.e., systolic and diastolic BP levels decreased by 4.4mm Hg & 2.5mm Hg respectively.

The other cardio vascular condition related to life style is at real fibrillation due to alcohol. Alcohol accounts for a l/3rd of new cases of atrial fibrillation. Acute/chronic alcohol injection can lead to AF. Due to release of catecholamine from adrenal medulla (Gregory Y.H. et al 1996).[7] Overall picture shown cardiovascular disease is the most important cause of death in alcoholics. Atherosclerosis of coronary arteries is responsible for almost all cases of CVD apart from other life styles associated with increased risk of CVD like diet, cigarette smoking, physical inactivity; Excessive alcohol consumption is also associated with a increased risk of CVD due to hypertension.

Epidemiological data indicates that moderate alcohol intake has a protective effect on CVD. Benefits from alcohol appear to be mediated mostly by an elevation in serum HDL cholesterol as well as by the effect of alcohol on platelets and fibrinolysis. There is a strong and inverse association between HDL cholesterol and the risk of CVD. The lower the concentration of HDL cholesterol the greater the risk of CVD. However, increased alcohol consumption is associated with an increased risk of CVD due to hypertension, haemorrhagic stroke or sudden arrhythmic death. For evaluation of the overall effect of life style on CVD the relative and attributable risks before adjustment with HT, hypercholesterolemia and glucose intolerance are important. Drug therapy should be administered to those under strong effect of genetic factors and those with intense degree of H.T. or hypercholestoralemia.

The time tested agent for primary management of hypertension coronary artery disease is Beta blockers. There are three types of

a) Non-cardio selective group
   Ex: - Metaprolol, Sotolol, timolo, nodolol
b) Cardio selective group
   Ex: - Metaprolol and Atenolol
c) Beta blockers with ISA (intensic sympathominetic activity)

Ex: - Pindolol and Beta adrenergic blocking drugs are the established agent for the treatment of hypertension in view of the postulated association between plasma lipids and coronary heart disease. It seems important to investigate the effect of Beta blockers on plasma lipids. The mechanisms of the changes in plasma lipid concentrations are observed with hypertension receiving treatment with Atenolol, Metaprolol, propranolol and oxpronolol in various studies.

Preventive measure for those with HT and hypercholesterolemia should preferable be taken throughout life. However modification of life style in most cases, particularly in younger generation can remarkably improve the health indexes.

OBJECTIVE OF STUDY: Beta adrenergic blockers have increasingly replaced diuretics as the first line of therapy in treatment of H.T. With availability of large No. of Beta blockers having differences in their pharmacological properties, the physician can now select the drug most suitable for a particular patient. There is increasing evidence that many selective and non-selective beta adrenoceptor blocking agents increase serum total cholesterol and TG and decrease HDL cholesterol Beneficial effects obtained by treatment of H.T by Beta blockers may be offset by their unfavorable effects on the plasma lipids. Although many studies were conducted to form definite guidelines in the risk Vs. benefit ratio, there is no clear cut consensus regarding this. Hence, it was considered to take up the study to see the effect of propranolol and atenolol on plasma lipids in alcoholic and non-alcoholic hypertensive patients in 8 weeks of therapy in our set of population.

The present study has been under taken with the following objectives: (1) Effect of alcohol on CVS, (2) To study the plasma lipoprotein pattern in relation to the therapy and selection of drug in alcoholics and non-alcoholics. (3) To draw a conclusion if possible between the two groups (4) To determine the changes in the treatment with respective alterations in lipid profile in relation to alcoholism.

MATERIAL/METHODS: The study was conducted on patients suffering from hypertension associated with alcoholism attending the medical O.P. of our hospital, K.G.H. Visakhapatnam. The investigations were carried out in the clinical laboratory of our hospital. Sixty male patients with Hypertension and History of alcohol drinking were taken into 2 groups. Each group consisted of 30 patients. One group was given Tab. - propranolol and the other group was given Tab. - Atenolol. Physical examination revealed mild to moderate hepatomegaly in 26 of the patients. "There was no clinical Jaundice in any one of them. Duration of alcohol drinking varied from 3 to 15 years and the quantity of alcohol intake varied between 80 to 150 gms/day. The lipid profile was estimated at the time of entry, 4 weeks and end of 8 weeks in both groups. All patients were examined clinically and B.P. was recorded at each visit. The routine laboratory parameters including Hb%, TC, DC, ESR, FBS, LFT (S.Bilirubin, SGOT, SGPT & SAP) renal function test (albumin, sugar, microscopic examination, urea, creatinine) and chest X-ray & ECG were taken at the time of entry. Blood Pressure readings were taken with random zero sphygmomanometer after resting the patient for 10-15 minutes on a couch in lying down position. The patients were given tab. - propranolol (40-80 mg) sustained release preparation and Tab. - Atenolol (50-100 mg) randomly.

SELECTION OF PATIENTS: Sixty male hypertensive patients attending the Medical OP department at K.G. Hospital, Visakhapatnam were selected for the present study. These sixty patients were divided into 2 groups of 30 each. They were untreated earlier and had established

high B. P. readings for 3 consecutive readings prior to the entry into the present study. The selected group includes:

a) All are male patients.
b) Patients between 35 to 75 years of age.
c) Patients who do not have cardiac or renal or pancreatic dis-function.
d) Patients without existing dyslipidemias.
e) Patients who are not taking other alternative medicines.
f) Patients not on multiple drug therapy.
g) Patients who do not have irregular dietary habits. .
h) Patients who are not having recent infections or major surgery.

LIPID PROFILE ESTIMATION: Lipid profile was done in all patients at the time of entry, 4 weeks & 8 weeks of therapy. Total cholesterol, TG, HDL are estimated by the enzymatic method, using auto pack kits. The patients were asked to come on 12 hours (overnight) fasting; patients were made comfortable and reassured before collecting 10ml of whole blood.

The data was analyzed by using student "t" test for paired values. Probability value was read from the available tables.

RESULTS & OBSERVATIONS: The study sample includes 60 male patients. They were divided into 2 groups consisting of 30 patients in each group.

Group A: This group consists of 30 patients with hypertension and history' of alcohol drinking. They were allocated to Tab. Atenolol. Among the 30 patients, 11 patients received 100 mgs per day and 19 received 50mgs per day.

Group B: This group includes 30 patients with hypertension and alcohol drinking. They are allocated Tab. propranolol. Out of 3*0 patients, 12 received 80mgs. Per day and 18 patients received 40mgs. Per day.

TYPE 'i	NORMAL VALUE
Cholesterol	130-250mg/dl
HDL Cholesterol	Risk if < 35
LDL Cholesterol	Risk if > 190
Triglycerides	10-165mg/dl.

Table 1: Biochemical normal values of lipids

AGE DISTRIBUTION	GROUP-A	GROUP - B
35-40	3	7
41-45	8	7
46-50	10	8
51-55	3	5
56-60	2	2
Above 60	4	1
Total	30	30

TABLE 2: DEMOGRAPHIC CHART AND DISTRIBUTION OF PATIENTS ACCORDING TO AGE GROUP

Duration of Illness: The duration of illness of both group is shown in Table III.

GROUP- A	14.2 ±5
GROUP - B	14.8 ± 12

TABLE 3: SHOWING THE DISTRIBUTION OF DURATION OF ILLNESS IN MONTHS

BLOOD PRESSURE RECORDINGS: The blood pressure recordings were taken at 0, 4 & 8 weeks of the study in both groups (Table - IV).

GROUP	DURATION IN WEEKS		
	0	4	8
A	170/105 ±40/10	143.5/91.8 ±25/30	125/85 ±30/10
B	160/105 ±20/15	140.8/105.8 ±22.5/13.3	126/84 ± 15/12.5

TABLE 4: SHOWING THE BP MM/HG RECORDINGS OF A & B GROUPS

The hypertensive efficacy of both Atenolol and propranolol was comparable. Both groups achieved good control of blood pressure by the end of 8 weeks. The mekn blood pressure at '0' week in group "A" was 170/105 ± 40/10mm Hg. *A* 4 weeks the mean blood pressure was 143.5/91.8 ± 25 30mm Hg. and at 8 weeks it was 125/85 ± 30/10.

In group 'B' the mean blood pressure reading at '0' week was 160/105 ± 20/15mm Hg. at '4' weeks it was 140.8/105.8 ± 22.5/13.3mm Hg. and at 8 weeks it was 126/84 ± 15/12.5mm Hg.

	0-4	4-8	0-8
SBP	30.2 (SD)	2.55 (SD)	4.8 (SD)
DBP	2.22 (SD)	1.17 (NSD)	7.6 (SD)

Table 5: Blood Pressure control in GROUP – A

	0-4	4-8	0-8
SBP	3.43(SD)	2.94(SD)	7.32(SD)
DBP	0.21(NSD)	6.54(SD)	5.79(SD)

TABLE 6: BLOOD PRESSURE CONTROL IN GROUP - B

SBP = Systolic Blood Pressure.
DBP = Diastolic Blood Pressure
SD = Significant Difference.
NSD = No Significant Difference.

CHANGES IN LIPID PROFILE: Alteration in lipid profile pattern in both the groups with Beta blocker therapy are shown in Table -VII.

TYPE DURATION	GROUP -A			GROUP B		
	0 Weeks	4 Weeks	8 Weeks	0 Weeks	4 Weeks	8 Weeks
TC	202.7±48	210±65	203±51	200.7±32	209.9±31.45	216.2±25
TGL	156±84	145±104	143±75	155.5±72	164.3±61.9	176.16±73.9
HDL	82±7	43 ±9	41.8 ±8.9	42.43±8.9	41.43±8.98	38.70±9
VLDL	30±16	30±21	29±21	32.3±51	33.26±12.48	33.8 ± 14.5
LDL	132.13±49.9	132.2 ±40	129.2±47.5	125.4±32.0	13.33±27.49	142.16±20.49

TABLE 7: SHOWING THE ALTERATION IN LIPID PROFILE IN GROUP - A AND GROUP-B

The mean total Cholesterol in Group A at 0, 4, & 8 weeks was 202.7 ± 48 mmg/dl, 210 ± 65 mg/dl and 203 ± 51 mg/dl.

The mean TGL of Group A at 0, 4 & 8 weeks of therapy was 156 ± 84, 145 ± 105 and 143 ± 75 mg'dl respectively.

The mean HDL of group A at 0, 4, & 8 weeks of therapy was 42 ± 14; 43 ± 9 and 41.8 ± 8.9 mg/dl respectively.

The mean VLDL of Group A at 0, 4, & 8 weeks of therapy was 30 ± 16; 30 ± 21 and 29 + 21 mg/dl respectively.

The mean LDL of Group A at 0, 4, & 8 weeks of therapy was 132.19 ± 49.9, 132.20 ± 40 and 129.2 ± 47.5 mg/dl respectively.

TYPE	0-4 't' 'p'	4-8 't' 'p'	0-8 't' 'p'
TC	0.4 (NSD)	0.4 (NSD)	0.02 (NSD)
TGL	0.44 (NSD)	0.083 (NSD)	0.62 (NSD)
HDL	0.37 (NSD)	0.51 (NSD)	0.07 (NSD)
VLDL	0.28 (NSD)	0.18 (NSD)	0.20 (NSD)
LDL	0.005 (NSD)	0.26 (NSD)	0.20 (NSD)

TABLE 8: Though there was little variation in individuals concentrations at 0-4, 4-8, 0-8 weeks of therapy with Atenolol, they are not statistically significant

Value tested at 0.05.

The mean TC of group B at 0, 4, & 8 weeks of therapy was 200.7±32, 209.0±31.45 and 216.2 ± 25mg/dl respectively.

The mean TG of group B at 0, 4, 8 weeks of therapy was 155 ± 72, 164.3 ± 61.95 and 176.16 ± 73.99 mg/dl respectively.

The mean HDL of group B at 0, 4 & 8 weeks of therapy was 42.43 ± 89, 41.43 ± 89, 41.43 ± 8.93 and 38.7 ± 9 mg/dl respectively.

The mean VLDL of group B at 0, 4, and 8 weeks of therapy was 32.3 ± 15. 33.26 ± 10.48 and 33.8 ± 14.5 mg/dl respectively.

The mean LDL of group B at 0, 4, & 8 weeks of therapy was 125.4 ≠ 32, 135.33 ± 27.49 and 142.16 ± 20.49 mg/dl respectively.

The variation in values for TC and LDL at 0 to 8 weeks level were statistically significant when tested for 0.05 level of 'p' value (Table. IX)

TYPE	0-4 't' 'p'	4-8 't' 'p'	0-8 't' 'p'
TC	1.10 (NSD)	0.83 (NSD)	2.05 (NSD)
TGL	0.49 (NSD)	0.66 (NSD)	1.09 (NSD)
HDL	0.42 (NSD)	1.15 (NSD)	1.58 (NSD)
VLDL	0.26 (NSD)	0.15 (NSD)	0.38 (NSD)
LDL	0.88 (NSD)	1.07 (NSD)	2.36 (SD)

TABLE 9

The atherogenic index was calculated with the following formula:

$$\text{Atherogenic Index (A. I.)} = \frac{\text{Total Cholesterol - HDL}}{\text{HDL}}$$

WEEKS	GROUP - A 't' 'p'	GROUP -B Y 't' 'p'
0-4	3.89 1.12(NSD)±2.4	3.76 0.43 (NSD)± 1.65
4-8	3.84 0.22(NSD)±1.28	4.17 0.21 (NSD)± 0.21
0-8	3.66 0.92(NSD)±1.50	4.62 0.18 (NSD)± 1.40
The values are in (Table-10) for Group-A and Group-B		

There was no significant difference in both groups observed at 0-4. 4-8, 0-8 weeks of therapy with both Atenolol and propranolol.

DISCUSSION: Several studies have shown that many cardio selective and non-cardio selective beta adrenergic blocking drugs increase serum total cholesterol and triglycerides levels and decrease HDL concentration. The high concentration of serum cholesterol and the ratio of HDL-C to total cholesterol are thou lit to reflect the atherogenicity of serum lipids.

In the contrary to the above finding, no change in total cholesterol was noted by several other authors (Tyagi. S. et al, 1990 and Famenbaum 1985).[8, 9]

Thus little doubt exists that some beta blockers may effect serum lipid in a possibly adverse manner, although acebutolol (Lehtonen A 1984)[10] and Pindolol which possess ISA have no untoward effects on serum lipids.

Tyagi S. et al (1990)[8] drew conclusion in his study among Indian population that treatment with propranolol mono therapy increased serum triglycerides, VLDL-C and decreased serum HDL-C and worsened atherogenic index. On the other hand Acebutolol a cardio selective drug showed no significant change in plasma lipids.

In the present study, the Atenolol group showed no lipid abnormalities during the treatment period. However, the propranolol group showed a significant adverse reaction in total cholesterol and LDL level at end of 8 weeks of propranolol therapy.

The enzyme Lipoprotein lipase degrades TG rich lipoprotein and transfers surface material to $HDL_?$ Converting HDL_3 to HDL_2 (Pataseh-Jr-R-eHri-4978). The enzyme Hepatic lipase degrades HDL_2 phospholipid and participates in the hepatic uptake of HDL_2 cholesterol. Inhibition of lipase would be activated through either a direct inhibitory action of adrenergic blocking agents themselves or secondary, unopposed alpha adrenergic stimulation. It is postulated that alpha adrenergic stimulation may have an important role in suppressing adipose tissue lipase. with secondary reduction in plasma HDL-C and an increase in triglycerides (Day JL et al 1982).[11] The

changes in serum lipase and adipose tissue lipoprotein lipase after the three week break in Sotalol (non cardio selective agent) treatment were analyzed.

It may be suggested that the decrease in HDL caused by beta blockers is not mediated by their effects on HDL. The clinical significance of the effects of propranolol and other similar drugs on lipoproteins is uncertain in terms of coronary risk. But any long medication which lowers HDL-C but not LDL-C in subjects who are already at risk of myocardial infarction on account of hypertension should be used with caution keeping in view the alcoholism as a risk factor. Total cholesterol to HDL cholesterol ratio or LDL-C to HDL -C ratio has been found to be the strongest lipid risk factor for clinical Ischaemic heart disease (Muller NE et al, 1987)[12] and progression of coronary atherosclerosis.

Apart from this, the intervening of the prevalence of the adverse effects resulted due to alcoholic risky lifestyle, also playing an important role in influencing the above parameters and finally throwing a fluctuation among the results of the daig effects with no exemption to p blockers.

Hence, clear clinical comparative studies on the effects of beta blockers with or without ISA on the incidence of coronary heart disease in view of the prevalence of the effected alcohol risky lifestyle would be of great value to the clinicians and in turn to the benefit of the patients.

Atherosclerosis and its complications account for half of all deaths in the industrialized world. It is usually associated with high serum cholesterol and hyperlipedemia. These have been difficult to explain.

LIPID PEROXIDES AND ATHEROSCLEROSIS: Lipid peroxides formed by peroxidation of unsaturated tally acids were first detected in a atheroclerosic human aorta more than 30 years ago and a subsequent study confirmed a strong positive coordination between the severity of aortic atherosclerosis and concentration of these compounds in the aortic wall. Several groups have since suggested that lipid peroxides may be important in the development of atherosclerosis. This might help to explain the frequent occurrence of disease in normolipideamic people. It was found that raised concentration of lipoperoxides have been found in diabetes, in patients with angiopathy in hyper lipidemia, essential hypertension and after myocardial infarction and stroke. Peroxidised lipids may be important in atlierogenesis and its complications and peroxide lipids may provide an index of the severity of atherosclerosis.

Lipid peroxideses are distributed throughout the lipoprotein fraction but are carried predominantly by low density lipoprotein, the density is most firmly linked with atherosclerosis. Moreover peroxidation of low density lipoprotein alters their biological proportions leading to increased up take by endothelial cells and macrophages, procoagulant activity in the blood and other functions relevant to atherosclerosis and its complications. Some authors have shown clinically significant rise in plasma concentration of these compounds in patients with angiographically proved occlusive arterial disease (both IHD and penfiaaj arterial disease). Lipid peroxidose may emerge as a useful index of severity *ei* disease or predictive information.

High serum cholesterol is considered a risk factor of coronary artery <£seas (CHD) and for production of atherosclerosis in the wall of arteries of any size m all part <^f the body, which may be responsible for angina and heart attack. Cerebrovascular accident and narrowing

peripheral arteries in legs, kidneys and eyes. Thus high cholesterol in the blood is responsible for possible cause of such disabling and fatal disease. Doctors and patients look for its cause and treatment. It may be mentioned that lowering of level of cholesterol is also associated with decreased incidence of atherosclerosis and coronary heart disease (CHD). Increased serum cholesterol particularly increased level of for density lipoprotein (LDL) and cholesterol which usually go together and are associated with CHD.

Cholesterol is a fat like lipid. It is a percussion of bile acids and steroid hormone. It circulates as a spherical particle. It contains both lipids and protein - lipoprotein. The level of cholesterol in the blood is dependant

1) on family inheritance,
2) on fat content of the diet,
3) obesity and
4) lack of exercise.

In the blood the lipoprotein are

a) Low density lipoprotein (LDL)
b) High density lipoprotein (HDL).

The L.DL is more atherogenic and is about 60-70 percent of total serum cholesterol. The HDL is about 20-30 percent of total cholesterol. The levels of these are inversely co-related to CHD. VLDL is largely composed to triglyceride. It is 10-15 percent of total serum cholesterol. The third component is very low density lipoprotein of triglyceride. It is 10 to 15 percent. Estimation of total cholesterol is less expensive test and does not require fasting state. It can be used for average routine test. LDL estimation has to be done fasting is more expensive and more accurate, is preferred to take clinical decisions for evidence of risk towards CHD.

Dietary factors and alcoholism have been implicated in the pathogenesis of diseases of liver. However, not enough data is available on dietary- intake and lipid changes among the cirrhotic groups with different etiologies like alcoholism and post- hepatitis cirrhosis.

SUMMARY: Thirty patients with hypertension in. association with excessive alcohol drinking were treated with Tab. Atenolol and another group of similar number of patients with same history was treated with Tab. propranolol. Lipoprotein alterations were measured in each group at 0, 4 & 8 weeks of therapy. It was concluded from the study that:

There was no alteration in lipid profile pattern in Atenolol treated group. The total cholesterol and LDL were significantly increased with propranolol. There was no alteration in TGL, VLDL & HDL cholesterol in propranolol treated group. There was no significant difference in atherogenic index in both the groups during the study period. Clinically observation pharmacotherapy regimes are being influenced by one of the other risky lifestyles like alcohol directly or indirectly.

The stress and strain caused by alcoholism through various pathogenic changes in the internal environment are having an impact on the prognostic and tlierapeutic values following therapy with p adrenoceptor blockers.

However, we are now in the era of life-style related diseases. The pathogens are present in individual's life styles and the living states. In such modern times, a new therapy of medical sciences and treatment need to be established. According to the theory, every member of the family changes his-her unhealthy lifestyles by learning of health through discussion with physicians and health medical care specialists. Thus, leading to a decrease in the risk of life style related diseases in the future. This in turn leads to an increase in the health potential of individual and community.

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