

Favourable Effect of Abana on Lipoprotein Profiles of Patients with Hypertension and Angina Pectoris

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ABSTRACT

Abana, a herbomineral drug, was found useful in controlling hypercholesterolemia. In the present series, the indigenous formulation, Abana, was given in normal as well as in cases of essential hypertension and angina pectoris. Abana reduces total cholesterol and triglycerides. A significant increasing trend was noticed in high-density lipoprotein cholesterol levels. It was found that Abana has the capacity to regulate hypercholesterolaemia and hypertriglyceridemia by regulating abnormal lipoprotein metabolism. Thus the use of, Abana may be advocated for the prevention and management of coronary heart disease.

INTRODUCTION

A large number of reports are available to show the significant role of lipoproteins in the incidence of coronary heart disease (CHD). It is currently proved that high levels of low density lipoprotein cholesterol (LDL-C) are deleterious. Similarly, a large number of reports have indicated that high density lipoprotein cholesterol level (HDL-C) is inversely related to the incidence of CHD^{8,7,4}. The role of very low density lipoprotein cholesterol (VLDL-C) as a risk factor is less certain. LDLs are the major cholesterol, carrying lipoproteins in the plasma. LDL receptors on liver cells are responsible for the removal of LDLs^{9,13}. The small amount of LDLs can be cleared by extrahepatic tissues. Ultimately, HDL-C may accept cholesterol from extrahepatic tissues and transfer it to VLDLs and LDLs. Finally, cholesterol carried on these latter lipoproteins is removed by the liver⁶.

In recent years the significance of plasma cholesterol levels in the occurrence of CHD has been repeatedly emphasised. The mechanisms for the synthesis, transport and catabolism of cholesterol are understood much better today than a mere decade ago. The significant contribution of Goldstein and Brown¹, which resulted in the discovery of cell surface receptors for LDL, is fundamental to our understanding of the need to control cholesterol levels^{1,6}. The finding of these workers provide a rational means for controlling cholesterol concentrations. Based on the Framingham Study, Castelli, *et al.*² demonstrated that the proportion of the total cholesterol (TC) carried by HDL-C is a consistent and important indicator of coronary risk in both sexes over the age of 49 years². The Lipid Research Clinics Coronary Primary Prevention Trial reported in 1984 that reduction of plasma cholesterol levels in turn reduces the frequency of CHD¹¹. Several large field surveys confirm the positive correlation between the concentration of plasma cholesterol and risk of CHD. The authentic data from the Framingham Heart Study and the Lipid Research Clinics Programme showed identical findings regarding the levels of cholesterol concentration and incidence of CHD^{2,5}. Several drugs have been advocated for the modification of different lipoprotein levels. Currently available drugs for the treatment of hypercholesterolemia have many side effects. Till now a positive benefit/risk ratio for the cholesterol-lowering drugs has been difficult to prove.

Several herbomineral drugs have been advocated for the prevention and management of CHD. The significance of *Terminalia arjuna* has recently been established in the management of ischaemic heart disease¹⁴. The drug Abana is a herbomineral compound advocated for the prevention and management of CHD. Abana contains *Terminalia arjuna*, *Withania somnifera*, *Tinospora*

cordifolia, Phyllanthus emblica, Terminalia chebula, Glycyrrhiza glabra, Asparagus racemosus, Boerhaavia diffusa, Centella asiatica, Convolvulus pluricaulis, Nardostachys jatamansi, Cyperus rotundus, Acorus calamus, Piper longum, Makaradwaja, etc., in different quantities.

Abana was given in apparently normal individuals and in diagnosed cases of essential hypertension and angina pectoris.

In order to study the clinical efficacy of Abana on lipoprotein metabolism, a careful clinical trial was carried out in selected normal, as well as essential hypertension and angina pectoris cases.

MATERIAL AND METHODS

Seventy-four diagnosed cases of essential hypertension and angina pectoris were included in our trial. Thirty-nine cases were suffering from essential hypertension and the remaining 35 from angina pectoris. To compare the results 30 apparently normal individuals were also selected. Only mild to moderate cases of essential hypertension were included. Total lipid profiles were carried out in all cases. Different fractions of lipoproteins were measured following the method developed by Laurell¹⁰.

After the initial investigations Abana was given to all three groups, two tablets t.i.d., continuously for 12 weeks. Placebo was introduced in the same manner to both normal and disease groups. All the investigations were repeated after 4 weeks to compare the results.

RESULTS

Abana showed a significant influence on TC levels (Table 1). In normal cases TC level showed a significant decreasing trend ($p < 0.05$). TC also significantly reduced in essential hypertension and angina pectoris cases. TG showed similar decreasing trends in the above two conditions (Table 2). In normal cases Abana enhanced the HDL-C level (Table 3). A low level of HDL-C was noticed in essential hypertension and angina pectoris cases, but after 12 weeks of Abana therapy a significant increasing trend in HDL-C was noticed in both the disease groups. LDL-C showed a high level in both essential hypertension and angina cases. After 12 weeks of Abana therapy LDL-C showed a significant decrease in the normal as well as in the diseased group (Table 4) VLDL-C also reduced in clinical conditions but in normal cases Abana could not show any change in VLDL-C level (Table 5).

Table 1: Changes in total cholesterol levels following oral administration of Abana in different groups (mean \pm SD mg/dl)

Group	Normal		Essential hypertension		Angina pectoris	
	Initial	12 weeks	Initial	12 weeks	Initial	12 weeks
Placebo	179.79 \pm 31.36	191.53 \pm 27.96	250.11 \pm 38.76	247.24 \pm 34.55	269.52 \pm 28.68	265.23 \pm 35.12
	n=12		n=12		n=12	
Treated	188.16* \pm 27.89	170.19* \pm 21.68	282.49 [•] \pm 45.42	240.72 [•] \pm 42.03	278.30 [■] \pm 48.78	233.78 [■] \pm 49.63
	n=18		n=27		n=23	
Placebo	p not significant		Treated * $p < 0.05$		[•] $p < 0.01$	[■] $p < 0.001$

Table 2: Changes in triglyceride levels following Abana treatment (mean ± SD mg/dl)						
Group	Normal		Essential hypertension		Angina pectoris	
	Initial	12 weeks	Initial	12 weeks	Initial	12 weeks
Placebo	138.10 ± 28.00	145.71 ± 33.00	208.02 ± 36.42	203.08 ± 36.92	243.15 ± 36.24	227.35 ± 44.22
	n=12		N=12		n=12	
Treated	143.49* ± 42.62	125.73* ± 35.43	235.59 [•] ± 25.27	211.91 [•] ± 33.41	236.00 [■] ± 45.47	203.94 [■] ± 32.77
	N=18		N=27		n=23	
Placebo <i>p</i> not significant Treated * <i>p</i> <0.05 [•] <i>p</i> <0.01 [■] <i>p</i> <0.02						

Table 3: Changes in HDL-C following Abana therapy (mean ± SD mg/dl)						
Group	Normal		Essential hypertension		Angina pectoris	
	Initial	12 weeks	Initial	12 weeks	Initial	12 weeks
Placebo	49.15 ± 7.00	48.94 ± 7.64	36.34 ± 8.61	37.42 ± 7.59	38.05 ± 8.15	40.25 ± 9.21
	n=12		N=12		N=12	
Treated	50.10* ± 7.55	54.11* ± 6.50	37.91 [•] ± 11.49	49.69 [•] ± 13.31	35.59 [■] ± 8.68	43.38 [■] ± 9.50
	n=18		N=27		N=23	
Placebo <i>p</i> not significant Treated * <i>p</i> <0.05 [•] <i>p</i> <0.01 [■] <i>p</i> <0.01						

Table 4: Changes in LDL-C in different clinical groups (mean ± SD mg/dl)						
Group	Normal		Essential hypertension		Angina pectoris	
	Initial	12 weeks	Initial	12 weeks	Initial	12 weeks
Placebo	92.35 ± 22.18	101.82 ± 25.31	158.45 ± 26.42	151.93 ± 20.20	182.62 ± 25.01	170.51 ± 31.72
	n=12		N=12		N=12	
Treated	98.89* ± 21.73	83.41* ± 18.78	170.96 [•] ± 35.39	136.37 [•] ± 36.32	180.75 [■] ± 47.25	138.66 [■] ± 46.45
	n=18		N=27		N=23	
Placebo <i>p</i> not significant Treated * <i>p</i> <0.05 [•] <i>p</i> <0.01 [■] <i>p</i> <0.01						

Table 5: Changes in VLDL-C levels after oral administration of Abana (mean ± SD mg/dl)						
Group	Normal		Essential hypertension		Angina pectoris	
	Initial	12 weeks	Initial	12 weeks	Initial	12 weeks
Placebo	35.29 ± 12.03	40.78 ± 13.49	53.32 ± 16.18	57.89 ± 18.45	65.76 ± 19.58	69.95 ± 19.59
	n=12		N=12		n=12	
Treated	40.23* ± 12.64	32.94* ± 11.56	73.60 [•] ± 23.43	53.67 [•] ± 27.80	63.17 [■] ± 19.51	48.29 [■] ± 16.08
	n=18		N=27		n=23	
Placebo <i>p</i> not significant Treated * <i>p</i> <0.05 [•] <i>p</i> <0.02 [■] <i>p</i> <0.01						

DISCUSSION

Abnormal plasma lipoprotein levels are closely related to atherosclerosis and coronary artery disease. Significant elevations in the concentration of any one of the lipoproteins may cause hypercholesterolemia. Therefore a proper understanding of the mechanism of lipoprotein metabolism is important in the prevention and management of CHD. Currently available drugs for the management of abnormal lipoproteins do not provide satisfactory results. From the present observation Abana shows a profound influence on the regulation of lipoprotein metabolism. Reduction in the serum cholesterol levels in essential hypertension and angina pectoris cases indicate the significance of Abana in the prevention of coronary heart disease. The currently available drugs have shown adverse side-effects and hence such drugs cannot be advocated for

continuous use. On the contrary Abana did not demonstrate any adverse side-effects even after continuous oral administration. Dubey *et al.*³ found significant decreasing trends in the levels of cholesterol and triglycerides (TG) following oral administration of Abana in CHD cases. It is possible that this herbomineral compound might regulate the abnormal elevations of TC and TG either by decreasing the production or by increasing the clearance of lipoproteins.

Castelli *et al.*² reported the significance of HDL-C in the prevention of atherogenesis. From the present study it is evident that Abana has increased the HDL-C in hypertension and angina pectoris cases. Hence, it can be concluded that Abana may prevent the future occurrence of CHD in likely victims.

Some of the ingredients of Abana like *Terminalia arjuna*, *Nardostachys jatamansi* and *Glycyrrhiza glabra* have been advocated in the indigenous system of medicine for the prevention of cardiovascular disorders. Singh *et al.*¹² observed a hypotensive and bradycardic effect of *Terminalia arjuna* in dogs. *Phyllanthus emblica* has been reported to increase body resistance against disease and decay. It might be possible that *Phyllanthus emblica* may also increase HDL-C levels.

LDL-C was also reduced in hypertension and angina pectoris cases. This further indicates the significance of Abana in the prevention of CHD. The relationship between high concentrations of LDL-C and the incidence of CHD has been established. The role of the LDL receptor in the liver is recognised as the crucial element in the regulation of cholesterol levels. Abana has shown a significant influence on LDL levels, though the changes are not consistent in normal cases. At present it is difficult to infer the modes of action of Abana in the reduction of LDL-C levels. It is possible that some of the components of Abana reduce LDL-C by the action on LDL receptors in the liver.

VLDL-C showed a decreasing trend in essential hypertension as well as in cases of angina pectoris after Abana therapy. The significance of VLDL-C as a risk factor has not been proved so far. It requires comprehensive study to establish a relationship between VLDL-C and the incidence of CHD.

In brief, an increasing trend of HDL levels indicates the antiatherogenic property of Abana. Hence Abana can be advocated as a protective measure against atherosclerosis, hypertension and coronary heart disease.

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