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CLINICAL STUDY

A COMPARATIVE CLINICAL STUDY ON EFFICACY OF BHAVITA AMALAKI CHOORNA AND SHUDDHA GUGGULU IN DYSLIPIDEMIA

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ABSTRACT

Background: Dyslipidemia, a lipoprotein metabolism disorder, contributes significantly to atherosclerosis leading to cardiovascular diseases, the major cause of morbidity and mortality in both developed as well as developing countries. This calls for an urgent interventional strategy. Dyslipidemia being a chronic condition has to be managed by easily available, safe and effective medicines. Based on these facts, the present study was planned. **Objectives:** To evaluate and compare the efficacy of *Bhavita Amalaki Choorna* and *Shuddha Guggulu* in the management of dyslipidemia. **Materials and Methods:** In this interventional, active controlled, open labeled study, a total of 33 dyslipidemic subjects were alternatively allocated to receive *Bhavita Amalaki Choorna* (BA; n=16) and *Shuddha Guggulu Vati* (SG; n=17) in a dosage of 3grams twice daily with *takra* (buttermilk) as *anupana* half an hour before food for 3 months out of which a total of 30 completed the study. The assessment criteria were recorded at baseline and after treatment; assessed for the efficacy of the interventions. **Results:** BA elicited statistically significant results in HDL and SG elicited statistically significant results in Total Cholesterol, LDL and Triglycerides. Both the drugs elicited significant results in various other objective parameters. There were statistically significant differences found only in HDL between the group. **Conclusion:** Both BA and SG can be effective prescriptions for the management of Dyslipidemia. *Amalaki* is having added benefit of being cost effective and easily available in most parts of India. Thus BA can be preferred over SG.

Key Words: Dyslipidemia; Bhavita Amalaki Choorna; Shuddha Guggulu; Emblica Officinalis

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INTRODUCTION:

Dyslipidemia is one of the lifestyle disorder which is identified as a potential risk factor for multitudes of diseases like Cardiovascular Diseases (CVD), Metabolic Syndrome and even hypertension[1]. Dyslipidemia is a disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency. These disorders may be manifested by elevation of the serum total cholesterol, low-density lipoprotein (LDL) cholesterol and triglyceride concentrations, and a decrease in the high-density lipoprotein (HDL) cholesterol concentration[2],which contributes significantly to atherosclerosis leading to cardiovascular diseases (CVD), the major cause of morbidity and mortality [3, 4]. The prevalence of the condition of dyslipidemia is increasing worldwide in alarming rate in both developed as well as developing countries, including India. An ICMR survey [5] report published in 2014 concluded that over 79% of general adult population covered in the survey has abnormality in at least one of the lipid parameters with no urban and rural difference. This shows the gravity of the problem created by dyslipidemia. This calls for an urgent interventional strategy which can be applied in mass, easily adaptable, natural and feasible.

Ayurveda gives prime importance to prevention than treating manifested disease [6]. *Amalaki* (*Emblika officinalis Gaertn.*) is a commonly available fruit, that can be used on daily basis safely *(nitya sevaneeya dravya)*[7]; it has *medohara* action[8], lekhana[9,10] action and animal studies have proved that it has hypocholesterolaemic as well as antioxidant properties[11]. It is one of the best rejuvenators for all ages. Its chemical constituents viz., ascorbic acid, carotene and bioflavonoids act on abnormal lipid levels in blood thereby either preventing atherosclerosis or scraping out atherosclerotic plaques [12]. *Bhavana* is the process in which a *dravadravya* (liquid substance) like *swarasa* (juice), *kashaya* (decoction), *ksheera* (milk), *taila* (oil), *ghruta* (ghee) or *jala* (water) is added to the choorna (powder) and is triturated till the all liquid portion is absorbed, here the optimum proportion of liquid is added that the choorna should sink and get *ardrata* (wet)[13]. Charakacharya says that by giving *bhavana* with their own *swarasa* or *swarasa* of other drugs which are having same *virya* (potency) enhances the potency of the main drug and thus exerts multiple actions[14]. Based on this concept it is assumed that *bhavana* to the powder of the *Amalaki* from its *swarasa* multiplies the benefit of the drug**. Earlier studies have also shown significant hypolipidemic action of *Shuddha Guggulu*[15].**

Dyslipidemia needs intervention to prevent the formation of atherosclerosis. Dyslipidemia being a chronic condition has to be managed

by means of long term consumption, with no adverse effect. The selected option must be economical, easily available, easy to administer. Some of the drugs are not suited for daily intake. The search for the suitable, safe and cost effective drug is the need of the hour. Based on the above facts, the present study on efficacy of *Bhavita Amalaki* in comparision with *Shuddha Guggulu* (*Commiphora mukul Stocks*.) in the management of dyslipidemia was planned.

MATERIALS AND METHODS:

Research design: It was an interventional, active controlled, open labeled clinical trial.

Study population: An accessible population of dyslipidemic subjects in and around Hassan, who were representative of target population, participated in the study.

Study sample: Previously known or freshly diagnosed subjects of dyslipidemia from in and around Hassan.

Sample size: A total of 33 subjects of dyslipidemia, willingly participating in the study after a preliminary screening were included in study.

Study setting: The study was conducted in Sri Dharmasthala Manjunatheshwara College of Ayurveda and Hospital (SDMA&H), Hassan, Karnataka, India, from March 2015 to February 2016.

Ethical considerations: The research protocol was approved by Institutional Ethics Committee (IEC) of SDM College of Ayurveda and Hospital, Hassan, Karnataka.(IEC No: SDMCAH / IEC / 141/13-14 date 05th April 2014).

Sample size: A total of 33 subjects of dyslipidemia, willingly participating in the study after a preliminary screening were included in study.

Diagnostic criteria: The diagnostic criteria for dyslipidemia as mentioned by AACE Lipid and Atherosclerosis Guidelines[16] was considered for the study i.e., Total cholesterol >200 mg / dl; LDL >100/dl; HDL for men <40 mg/dl and females <50mg/dl; Triglycerides >150 mg/dl.

Inclusion Criteria: Subjects with age group of 20 to 60 years, irrespective of the gender having at least one of the diagnostic criteria and who are willing to sign informed consent.

Exclusion Criteria: Subjects having history of serious cardiac disorders like Myocardial Infarction, cardiac failure, uncontrolled hypertension, poorly controlled or newly diagnosed diabetes mellitus, subjects taking some new therapy or recently adjusted therapy, other systemic disorder viz., thyroid disorder, renal disorder, cholelithiasis and PCOS., those on drugs affecting the lipid levels like OC pills, statins, pregnant females and lactating mother.

Procurement and Method of Preparation of medicines: Raw *Guggulu* (*Commiphora mukul Stocks*.) and *Amalaki* (*Embilica Officinalis*

Gaertn.) were purchased from SDM Ayurveda Pharmacy, Kuthpady, Udupi and authenticated in the department of Dravyaguna, SDM College of Ayurved, Hassan. Preparations of both the medicines were done in SDM teaching Pharmacy, Hassan.

Method of preparation of *kashaya* for *bhavana*: Dried *Amalaki* was taken and pounded in *khalva yantra* (mortar & pestle) to make coarse powder. Then *kashaya* is prepared by adding 16 parts of water to 1 part of drug on a gas stove and *mandaagni* (low flame) was maintained till the water gets reduced to 1/4th. It was then filtered through a sieve and kept in a vessel. As preparation of *swarasa* from fresh *Amalaki* was very difficult and costly, *chaturthamsha kashaya* (1/4th part reduced decoction) *bhavana* is opted.

Preparation of BA: Raw *Amalaki* was cleaned and powdered. The quantity of *kashaya* sufficient to dip the amount of *choorna* was taken. *Amalaki choorna* was given three *bhavana* of *Amalaki chaturthamsha kashaya*. It was then powdered, measured (6 gms x 15 days = 90 gms) and packed in polyethylene zip lock covers with a spoon which measured 3 gms of powder.

Preparation of *Triphala Kashaya*: Dried raw *Triphala* was taken and pounded in *khalva yantra* to make coarse powder. Then *kashaya* is prepared by adding 16 parts of water to 1 part of drug on a gas stove and *mandaagni* was maintained till the water gets reduced to 1/8th. It was then filtered through a sieve and kept in a vessel.

Method of preparation of SG: Raw *Guggulu* was broken into small pieces and bundled in a piece of the cloth and boiled in *dola yantra* containing *triphala kashaya*. The boiling was continued till the *Guggulu* became soft mass. Then it was taken out of the cloth and spread over a smooth wooden board smeared with *ghrita*. By pressing with fingers, the sand and other impurities were removed. It is then taken out and reheated till it became semisolid consistency and then pounded in a stone mortar. This was then rolled into pills of 1 gm each and dried under sun. It was packed in a polyethylene zip lock covers containing 90 pills in each.

Treatment Methodology and schedule:

Diagnosed cases of dyslipidemia willing to undergo study were screened initially. Those who fulfilled the inclusive criteria were included for the study. Subjects were alternatively divided into trial group (BA group) and control group (SG group). Methodology of treatment for each group is summarized in table 1.

Table 1. Methodology of interventions in Trial group and Control group

Trial Group (BA group)	**Control Group (SG group)**
Administered with *Bhavita Amalaki Choorna* in a dosage of 3 gm twice daily with *takra* (buttermilk) as *anupana* half an hour before food for 3 months.	Administered with *Shuddha Guggulu* (in the form of *vati*) in a dosage of 3gm twice daily with *takra* as *anupana* half an hour before food for 3 months.

Subjects from both the groups were assessed before and on completion of study. Follow up after the intervention was not done. No specific diet or exercise pattern was advised during the study period. Subjects were only advised to avoid the excess usage of oils in the dietary articles.

Assessment Criteria:

Both the groups were assessed before and after the intervention on the basis of biochemical parameters with regards to Total cholesterol (mg/dl), LDL-C (mg/dl), HDL-C (mg/dl) and Triglycerides (mg/dl); objective parameters included weight in kg, BMI in kg/m^2, abdominal circumference, right & left mid-arm circumference, right & left mid-thigh circumference in centimeters and waist-hip ratio.

Statistical Analysis:

Statistical analysis of the result of 30 subjects who completed the study was done using SPSS VER. 20**.** Paired t-test was done to analyze the results within the group**.** Unpaired t-test (Independent sample t-test) with Confidence Interval 95% was done to analyze the results between the groups**.** The obtained results were interpreted in the statistical terms as, not significant (NS): $p > 0.05$**,** significant (S): $p < 0.05$.

OBSERVATIONS AND RESULT:

A total of 33 subjects volunteered to participate who fulfilled both diagnostic and inclusion criteria, also gave a written consent to be a part of study. A total of 30 subjects completed the study rests were dropped out due to various personal reasons. The BA group involved a total of 16 subjects among which 15 completed the intervention; SG group had 17 subjects out of which a total of 15 completed it. Study had 51.5% (n=17) of males and 48.5% (n=16) of females which indicating almost equal gender distribution in dyslipidemia. The condition was dominated by subjects in age group 20 to 29 (51.5%, n=17) followed by age group 30 to 39 and 50 to 59 (18.2 %, n=6 in each of them) indicating the shift of condition from older age group to younger age group. The nature of work based on physical activity involved majority of sedentary workers (54.5%, n=18) showing sedentary life as a risk factor for dyslipidemia. Majority of the subjects were *Kapha* dominant *prakriti* viz.,

Kapha-Vata prakriti (45.5%, n=15) and *Kapha-Pitta prakriti* (33.3%, n=11). The condition dyslipidemia invariably involves *Kapha.* Hence it can be understood that subjects who have dominance of *Kapha prakriti* will always be at risk of dyslipidemia. Majority of the subjects belonged to urban area (66.7%). The subjects presented with very few clinical signs i.e., Xanthealsma palpebrum in one; Xanthomata of elbow in one subject; Majority of them were asymptomatic. This indicates that dyslipidemia is an iceberg condition. Measurement of body mass index revealed overweight (42.4%, n=14) and obesity (36.4%, n=12) in majority of the subjects. Among the 17 males under study, waist hip ratio was increased in 10 males (58.8%); among 16 females it was increased in 15 females (93.8%). These findings position females at more risk than males[17]. Single lipid abnormality was found in 21.2% (n=7) of subjects, rest all had two or more lipid abnormality. Triglyceride abnormality was seen in 90.9% (n=30) of the subjects followed by abnormality of low density lipoprotein (72.7%, n=24). The results obtained in response to therapy within and between BA group and SG group for lipid profile and anthropometric measurements [table 2, table 3, table 4, table 5, table 6, table 7] are summarized. It was seen that BA group had statistically significant results in HDL and SG group showed statistically significant results in Total Cholesterol, LDL and Triglycerides with p value <0.05. When tested between the groups, there were statistically significant differences found only in HDL. On anthropometric measurement both BA group and SG group showed significant (p-value < 0.05) result in weight, BMI, upper mid arm circumference, mid thigh circumference and only SG group demonstrated significant (p <0.05) reduction in abdominal circumference. However, there was no statistically significant difference found in any of the anthropometric measurements between the groups.

Flow Diagram:

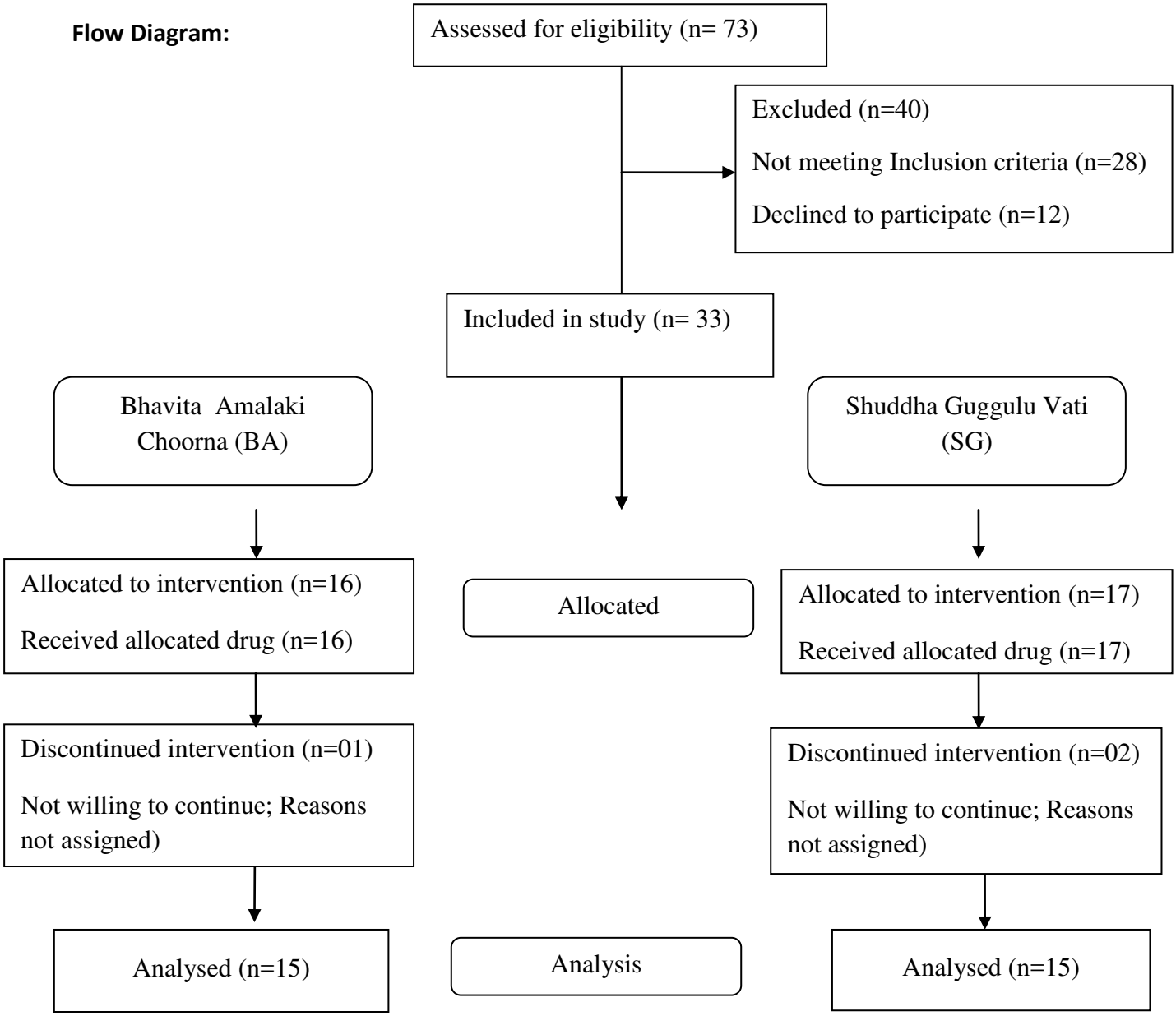

Table 2. Effect of *Bhavita Amalaki Choorna* on Lipid Profile

Parameters	Mean (mg/dl)		Diff. in Mean (BT-AT)	Paired 't' test				95% Confidence Interval of the Difference		Remarks
				S.D.	S.E.M.	t-value	p-value	Lower	Upper	
TC	BT	218.6	7.26	25.13	6.488	1.12	0.28	-6.650	21.183	NS
	AT	211.4								
HDL-C	BT	44.2	-4.50	5.65	1.459	-3.08	0.008	-7.629	-1.370	S

	AT	48.7								
LDL-C	BT	136.3	5.39	24.92	6.436	0.83	0.416	-8.408	19.200	NS
	AT	130.9								
TG	BT	180.1	21.96	45.18	11.666	1.88	0.081	-3.061	46.981	NS
	AT	158.1								

TC: Total Cholesterol, LDL: Low Density Lipoprotein-Cholesterol, HDL: High Density Lipoprotein-Cholesterol, TG: Triglyceride, BT: Before Treatment, AT: After Treatment, SD: Standard Deviation, SEM: Standard Error Mean, S: Significant, NS: Not Significant

Table 3. Effect of *Bhavita Amalaki Choorna* on objective parameters

Parameters	**Mean score**		**Diff in Mean (BT-AT)**	**Paired 't' Test**				**95% Confidence Interval of the Difference**		**Remarks**
				S.D.	**S.E.M.**	**'t' value**	**'p' value**	**Lower**	**Upper**	
Weight (Kg)	BT	73.18	1.16	0.605	0.156	7.455	0.000	0.830	1.501	S
	AT	72.01								
BMI (Kg/m 2)	BT	28.13	0.45	0.191	0.494	9.205	0.000	0.348	0.560	S
	AT	27.68								
Abdominal circumference (cm)	BT	91.23	1.63	2.255	0.582	2.804	0.14	0.384	2.882	NS
	AT	89.60								
Waist- Hip ratio	BT	0.92	0.001	0.018	0.004	0.274	0.788	-0.009	0.011	NS
	AT	0.91								
Right mid	BT	29.7	1.066	1.09	0.284	3.756	0.002	0.457	1.675	S

upper arm circumference (cm)		6								
	AT	28.70								
Left mid upper arm circumference (cm)	BT	29.66	0.866	1.04	0.269	3.218	0.006	0.289	1.444	S
	AT	28.80								
Right mid thigh circumference	BT	50.40	1.73	2.02	0.523	3.314	0.005	0.611	2.855	S
	AT	48.66								
Left mid thigh circumference	BT	50.06	1.56	1.74	0.449	3.485	0.004	0.602	2.530	S
	AT	48.5								

BMI: Body Mass Index, BT: Before Treatment, AT: After Treatment, SD: Standard Deviation, SEM: Standard Error Mean, S: Significant, NS: Not Significant, BT: Before Treatment, AT: After Treatment, SD: Standard Deviation, SEM: Standard Error Mean, S: Significant

Table 4. Effect of *Shuddha Guggulu Vati* on Lipid Profile

Parameters	**Mean (mg/dl)**		**Diff. in Mean (BT-AT)**	**Paired 't' test**				**95% Confidence Interval of the Difference**		**Remarks**
				S.D.	**S.E.M.**	**t-value**	**p-value**	**Lower**	**Upper**	
TC	BT	213.8	21.03	25.64	6.620	3.177	0.007	6.834	35.23	S
	AT	192.8								
HDL-C	BT	48.1	0.89	7.33	1.893	4.953	0.644	-3.166	4.953	NS
	AT	47.2								
LDL-C	BT	124.2	12.52	20.80	5.371	2.332	0.035	1.005	24.04	S

	AT	111.7								
TG	BT	207.6	38.41	30.42	7.855	4.890	<0.001	21.56	55.26	S
	AT	169.2								

TC: Total Cholesterol, LDL: Low Density Lipoprotein-Cholesterol, HDL: High Density Lipoprotein-Cholesterol, TG: Triglyceride, BT: Before Treatment, AT: After Treatment, SD: Standard Deviation, SEM: Standard Error Mean, S: Significant, NS: Not Significant

Table 5. Effect of *Shuddha Guggulu Vati* on objective parameters

Parameters	**Mean score**		**Diff in Mean (BT-AT)**	**Paired 't' Test**				**95% Confidence Interval of the Difference**		**Remarks**
				S.D.	**S.E.M.**	**'t' value**	**'p' value**	**Lower**	**Upper**	
Weight (Kg)	BT	73.34	1.537	0.869	0.224	6.844	<0.001	1.055	2.019	S
	AT	71.80								
BMI (Kg/m^2)	BT	28.34	0.59	0.439	0.113	5.219	<0.001	0.348	0.835	S
	AT	27.75								
Abdominal circumference (cm)	BT	94.53	1.70	1.048	0.270	6.278	<0.001	1.119	2.280	S
	AT	92.83								
Waist- Hip ratio	BT	0.93	0.018	0.039	0.010	1.749	0.102	-0.004	0.0400	NS
	AT	0.91								
Right mid upper arm circumference (cm)	BT	29.78	0.786	0.710	0.183	4.291	0.001	0.393	1.179	S
	AT	29.00								

Left mid upper arm circumference (cm)	BT	29.66	0.693	0.753	0.194	3.564	0.003	0.276	1.110	S
	AT	28.96								
Right mid thigh circumference	BT	51.00	0.866	1.07	0.278	3.117	0.008	0.270	1.463	S
	AT	50.13								
Left mid thigh circumference	BT	50.66	0.766	1.193	0.308	2.488	0.026	0.105	1.427	S
	AT	49.90								

BMI: Body Mass Index, BT: Before Treatment, AT: After Treatment, SD: Standard Deviation, SEM: Standard Error Mean, S: Significant, NS: Not Significant, BT: Before Treatment, AT: After Treatment, SD: Standard Deviation, SEM: Standard Error Mean, S: Significant

Table 6. Effect of trial group in comparision with control group on Lipid Profile

Parameters	**Group**	**No.**	**Mean (BT-AT)**	**Diff in Mean (BT-AT)**	**Unpaired 't' Test**			**95% Confidence Interval of the Difference**		**Remarks**
					S.Err. Diff	**'t' value**	**'p' value**	**Lower**	**Upper**	
TC	BA group	15	-7.266	13.76	9.270	1.485	0.149	-5.222	32.755	NS
	SG group	15	-21.033							
HDL-C	BA group	15	4.500	5.393	2.390	2.257	0.032	0.497	10.289	S
	SG group	15	-0.893							
LDL-C	BA group	15	-5.396	7.130	8.383	0.851	0.402	-10.04	24.302	NS
	SG group	15	-12.526							
TG	BA group	15	-21.96	16.4	14.064	1.170	0.252	-12.355	45.262	NS

	SG group	15	-38.41	53						

BA: Bhavita Amalaki Choorna ,SG: Shuddha Guggulu Vati, TC: Total Cholesterol, LDL: Low Density Lipoprotein-Cholesterol, HDL: High Density Lipoprotein-Cholesterol, TG: Triglyceride, BT: Before Treatment, AT: After Treatment, SD: Standard Deviation, SEM: Standard Error Mean, S: Significant, NS: Not Significant

Table 7. Effect of trial group in comparision with control group on objective parameters

Parameter	**Group**	**No.**	**Mean (BT-AT)**	**Diff in Mean (BT-AT)**	**Unpaired 't' Test**			**95% Confidence Interval of the Difference**		**Remarks**
					S.Err .Diff	**'t' value**	**'p' value**	**Lower**	**Upper**	
Weight (kg)	BA group	15	-1.166	0.371	0.273	1.357	0.186	-0.189	0.932	NS
	SG group	15	-1.537							
BMI (Kg/m 2)	BA group	15	-0.454	0.137	0.123	1.110	0.276	-0.116	0.390	NS
	SG group	15	-0.592							
Abdominal circumference (cm)	BA group	15	-1.633	0.666	0.642	0.104	0.918	-1.249	1.382	NS
	SG group	15	-1.700							
Waist-Hip ratio	BA group	15	-0.001	0.166	0.011	0.104	0.918	-0.006	0.039	NS
	SG group	15	-0.018							
Right mid upper arm circumference (cm)	BA group	15	-1.066	-0.280	0.338	-0.828	0.414	-0.972	0.412	NS
	SG group	15	-0.786							
Left mid	BA group	15	-0.866	-	0.33	-	0.606	-0.853	0.507	NS

upper arm circumference (cm)	SG group	15	-0.693	0.173	2	0.522				
Right mid thigh circumference	BA group	15	-1.733	-0.866	0.592	-1.463	0.155	-2.079	0.346	NS
	SG group	15	-0.866							
Left mid thigh circumference	BA group	15	-1.566	-0.800	0.544	-1.468	0.153	-1.916	0.316	NS
	SG group	15	-0.766							

BA: Bhavita Amalaki Choorna ,SG: Shuddha Guggulu Vati, BMI: Body Mass Index, SD: Standard Deviation, SEM: Standard Error Mean, NS: Not Significant

DISCUSSION:

The present study demonstrated the utility of *Bhavita Amalaki Choorna* in comparison with *Shuddha Guggulu Vati* in dyslipidemia. There are various studies conducted on the hypolidemic, antihyperlipidemic, antiatherogenic effect of *Amalaki* in various forms. Administration of alcoholic extract of *E. Officinalis* at a dose of 1g/kg/day to Albino Rats fed with high fat diet showed significant decrease in all the lipid parameters with a significant rise in the value of HDL (Lama a et al.,2013) [18]. A clinical study by Antony B et al(2008), found that aqueous extract of *Amlalaki* fruits at the dose of 500 and 1000 mg/day for 6 months in hypercholesterolemic humans attenuates the level of TC and lipid profile by inhibiting HMG CoA reductase and squalene epoxidase[19]. In addition the study done by Anila L. et al(2000), has been observed that flavonoids from *E.officinalis* lowered the lipid levels in serum and tissues of rats by inhibiting HMG CoA reductase and increased degradation and elimination of cholesterol in hyperlipidemic rats[20]. Also Takayo et al (2010) conducted an animal study

to evaluate hypolipidemic and antioxidant effects of *Amlalaki* (*Emblica officinalis Gaertn*.). Ethyl acetate extract of *Amla*laki (amla) has been reported to show its hypolidemic potential in age-related hyperlipidaemia at the dose of 40 or 10 mg/kg body weight per day for 100 days in young rats aged 2 months and aged rats aged 10 months by up regulating the levels of Peroxisome Proliferator activated receptor- α (protein which regulates the transcription of genes involved in lipid and cholesterol metabolism)[21]. An experimental study by Antony B et al(2006), in white rabbits showed that feeding of *Amalaki* extract (10 mg and 20 mg/kg) for 4 months reversed dyslipidemia and atheromatous plaques; the lumen of the aorta became normal as in the normal control group. They opined that reversal of dyslipidemia and atheromatous plaques achieved by *Amalaki* extract seems to be brought about by a number of factors, such as its ability to prevent low-density lipoprotein oxidation, its antioxidant action, besides decreasing synthesis of cholesterol by inhibiting 3-hydroxy-3-methylglutaryl-Coenzyme A reductase activity and elevating high-density lipoprotein level to enhance reverse cholesterol transport[22] Jeevanagi et al (2013), conducted an experimental study to evaluate hypolipedimic and anti-atherogenic activity of fruit of *Amalaki* in high fat fed albino rats. They concluded that administration of *Amalaki Choorna* 540mg/kg/day along with high fat diet (10ml/kg/day) for 8 weeks in comparison with Atorvastatin at a dose of 7.2mg/kg administered for 8 weeks, showed a significant decrease in all the lipid parameters ($p < 0.01$) with a significant rise in the value of serum HDL ($p < 0.01$) in both groups[23]. Also, in another study by Rupal A et al, administration of *Amalaki* powder (2.5, 5 and 10 gm%) to fluoride induced three month old male Albino rats (*Charles Foster*; 200- 250 gm bw) through diet significantly reduced plasma and hepatic lipid levels, tissue lipid peroxidation and increased plasma HDL-C and fecal cholesterol levels. Both hepatic HMG- CoA reductase activity and the bile acid (hepatic and fecal) production increased[24]. Similarly, work by Mathura R et al (1996), fresh juice of *Amalaki* (*E. officinalis*) at a dose of 5 ml/kg for 60 days in cholesterol-fed rabbits reduced Serum cholesterol, TG, phospholipid and LDL levels by 82%, 66%, 77% and 90%, respectively by altering the absorption of cholesterol and phospholipids and by increasing their excretion[25].

The hypolipidemic effect of gugulipid and guggulsterone has also been consistently demonstrated in various animal species, including rat, mouse, rabbit (Satyavati 1966; Satyavati et al. 1969), chicken (Baldwa et al.

1981), domestic pig (Khanna et al. 1969), dog and monkey (Dixit et al. 1980). Most scientific evidence suggested that guggulipid elicits significant reductions in serum total cholesterol, low-density lipoprotein (LDL), and triglycerides, as well as elevations in high-density lipoprotein (HDL)[26,27,28]. An experimental study done by Lata et al(1991), showed administration of ethyl acetate extract of C. mukul 200 mg/kg to albino rats weighing 300 + 15gm confer significant protection against atherogenic diet induced atherosclerosis by significantly preventing rise in serum cholesterol and serum triglyceride level [29]. Similarly, Shah R et al (2012) conducted experimentation. In that daily administration of guggulsterone (25 mg/kg b.w.) in rat models for 10 days lowered levels of serum cholesterol and triglycerides by 27% and 32%, respectively. In this study, the effect of guggulsterone on LDL-receptor binding activity was also investigated. Results showed that there was a significant increase in LDL binding to the cell membranes of hepatocytes in guggulsterone-treated rats[30,31]. A study of lipid lowering activity in triton and cholesterol-fed hyperlipaemic rat models was undertaken by Chander r et al (1996), examined the serum lipid levels in these rat models. Serum lipids were significantly lowered by guggulsterone (50mg/kg b.w.) in triton WR 1339-fed rats. In cholesterol-fed rats (25 mg/kg b.w.), simultaneous feeding of guggulsterone (5 mg/kg b.w.) decreased serum levels of lipids and apoprotein levels of VLDL and LDL. Activation of plasma and liver lipolytic enzymes stimulates the receptor mediated breakdown of LDL. Guggulsterone inhibits biosynthesis of cholesterol in liver and induces the excretion of bile acids in faeces and the activity of plasma lecithin: cholesterol acyltransferase [30,32]. Similarly in another study by Batra S.et al (2000), gugulipid was used as a positive control agent to evaluate the antioxidant, cardioprotective, and hypolipidemic activities of a series of synthetic compounds. Rats received gugulipid orally at a dose of 50 mg/kg for 30 days; gugulipid significantly decreased serum total cholesterol (35%) and lipid peroxide levels (57%). Hepatic microsomal lipid peroxidation was also significantly reduced by gugulipid. In addition, gugulipid significantly reversed the cardiac damage and biochemical changes induced by isoproterenol[33,34]. Clinical study conducted in 61 patients of hypercholesterolemia subjected to low-fat diet containing fruit- and vegetable-enrichment for 12 weeks prior to the treatment, then they received gugulipid 50 mg twice daily for 24 weeks followed by a 12-week washout period. Gugulipid reduced total cholesterol levels by 11.7%, LDL by 12.5% and triglycerides by 12%. HDL increase was not

statistically significant. After a 12-week washout period subjects treated with gugulipid exhibited substantial increases in total cholesterol by 6.5%, LDL by 6.6%, and triglycerides by 7.7%[33,35].

Probable mode of action of *Bhavita Amalaki Choorna*:

The probable action in reducing serum lipids are attributed to its antioxidant properties, which are derived from a high Vitamin C content and also from a large amount of tannin compounds viz., Emblicanin A and B, flavonoids that are also found potent antioxidants. It has been observed that flavonoids from *E.officinaisl* is lowered the lipid levels in serum and tissues of rats by inhibiting HMG CoA reductase and increased degradation and elimination of cholesterol in hyperlipidemic rats[20]. *Embilica Officinalis* is containing phenolic groups like Tannins, Gallic acid which act like statin. Like statin, *Embilica Officinalis* inhibits HMG CoA reductase activity. Ellgitannins and Ellagic acid obtained on hydrolysis of tannins inhibits epoxidase enzyme, a rate limiting enzyme of cholesterol biosynthesis[19]. Presently observed high levels of HDL in BA group could be related to the ascorbic acid and the flavonoid content of *Amalaki*, as ascorbic acid and flavonoids have been reported to increase the HDL- C content[33]. Supportive to this, one of the study shown that after *bhavana* to *Amalak*i results in a 3-fold increase of ascorbic acid[36]. The fruit yields good amount of dietary fibers. Dietary fibers are reported to increase the excretion of cholesterol by interfering with enterohepatic circulation of cholesterol[37,38,39]. Phytosterols are known to inhibit cholesterol absorption from the intestine due to their greater hydrophobicity and greater affinity for micelles than cholesterol itself and displace the intestinal cholesterol[40]. Saponins present in *Amalaki* are capable of precipitating cholesterol from micelles and interfere with enterohepatic circulation of bile acids, making them unavailable for intestinal absorption, leading to a reduction in plasma and hepatic cholesterol levels[41]. Though there was statistically significant increase found only in HDL Cholesterol, a mean reduction was observed in LDL, Total Cholesterol and Triglyceride compared to the baseline value. Probably by increasing dose of the drug may yield statistically significant result in all lipid parameters.

Probable mode of action of *Shudha Guggulu Vati* :

The possible mode of action in reduction of Total Cholesterol, Triglycerides, LDL could be related to the Guggulsterone (GS) - the bioactive constituent of guggul which is identified as a responsible factor for guggul's therapeutic effects[42]. Studies in vitro and in vivo showed that GS E& Z gum guggul may

inhibits cholesterol biosynthesis, prevents adrenaline induced free fatty acids release from fat cells (antilipolytic activity). It has been established that guggulsterone is an antagonist at farnesoid x receptor (FXR), a key transcriptional regulator for the maintenance of cholesterol and bile acid homeostasis[33.43]. The FXR antagonism by guggulsterone has been proposed as a mechanism for its hypolipidemic effect[43,44]. Based on another systemic study on gum guggul, it has been found that the ethyl fraction of gum shows anti-inflammatory effects and antioxidant effects in vitro[45]. Antioxidant effect prevent oxidation or may delay the oxidation thus prevent atherosclerosis.

Adverse effects: No adverse effects were noticed in both the trial as well as control group.

Palatability and drug compliance: Majority had no problem in consuming the drugs. Two subjects said *Bhavita Amalaki Choorna* was initially difficult due to its sourness and later they got adapted. The *Shuddha guggulu* which was in the form of vati of one gram was difficult to swallow as per one subject and was suggested to break it before consumption.

Specific benefits mentioned by subjects voluntarily in study group

In this present study, constipation (n=1), hair fall (n=1) was reduced and sense of well being (n=2) was told voluntarily by the subjects.

CONCLUSION:

Dyslipidemia is an iceberg disease as it does not exhibit any symptoms most of the time and hence is less reported. It should be managed by means long and safe effective medicines. Trial group showed mean reduction in Total Cholesterol (7.26 mg/dl, $p>0.05$), LDL Cholesterol (5.39 mg/dl, $p>0.05$), Triglycerides (21.96 mg/dl, $p>0.05$) and mean increase in HDL Cholesterol (4.50 mg/dl, $p<0.05$) in comparison with baseline values. There was no significant difference found in lipid parameters between the group except HDL-C ($p<0.05$). Hence on comparison *Bhavita Amalaki Choorna* can be effective prescription for the management of Dyslipidemia. *Amalaki* is cost effective and easily available in most parts of India. These suggest *Bhavita Amalaki Choorna* can be preferred over *Shuddha Guggulu.*

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