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A comparative clinical study of *Asanadi Ghanavati* and *Gomutra Haritaki* in *Kapha Medo Margavarana* (dyslipidemia)

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Abstract

Background: Dyslipidemia is a disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency and it can be understood in the parlance of the closest conditions in Ayurveda, viz. *Kapha Medo Margavarana* (dyslipidemia), *Atisthaulya* (obesity) or *Meda Roga* and *Prameha*. *Asanadi Ghanavati* (AG) is a modified presentation of *Asanadi Gana* drugs referred in Ashtanga Hridaya and *Gomutra Haritaki* (GH) is described in Charaka Samhita under *Shotha Chikitsa* and Ashtanga Hridaya in *Arsha Chikitsa*. **Aim:** To evaluate and compare the clinical effect of AG and GH in *Kapha Medo Margavarana*. **Materials and Methods:** Patients with the high lipid profile were selected and randomly divided into two groups. In Group A (n = 30), patients were administered with tablet of AG 1 g (500 mg each) thrice a day for 8 weeks and in Group B (n = 30), tablet of GH in similar dose and duration. Effect of therapy was assessed by body circumference, Body Mass Index (BMI), cardinal symptoms like *Anga-Gaurava*, *Bharavriddhi,* etc., and lipid profile parameters. **Result:** AG decreased the serum cholesterol by 7.12%, Serum Triglyceride (S. TG) by 7.72%, Serum Low Density Lipoprotein (S. LDL) by 11.68%, Serum Very Low Density Lipoprotein (S. VLDL) by 7.73%, and had increased Serum High Density Lipoprotein (S. HDL) by 9.52%, with moderate improvement in 14.81% and mild improvement in 70.37% of patients. The GH decreased the serum cholesterol by 6.31%, S. TG by 9.61%, S. LDL by 12.55%, serum VLDL by 8.99%, and increased S. HDL by 10.52% with moderate improvement in 3.70%, and mild improvement in 74.07% patients. **Conclusion:** AG and GH are suggested to be used in *Kleda Bahul Samprapti Janya Vyadhi* and *Ama Bahul Samprapti Janya Vyadhi* respectively.

Key words: *Asanadi Ghanavati*, dyslipidemia, *Gomutra Haritaki*, *Kapha Medo Margavarana*

Introduction

In the modern era, sedentary life-style and drastic changes in food pattern may lead to diseases such as dyslipidemia, type-II Diabetes Mellitus (DM), hypertension, and obesity, which are closely linked with each other and often co-exist in individual making the syndrome more complex and difficult to manage. Dyslipidemia stands as the most firmly established and the modifiable risk factor for cardio- and cerebro-vascular accidents, due to the presence of abnormal level of cholesterol, which is a causative factor for atherosclerosis. According to National Commission on Macroeconomics and Health, there would be around 62 million patients with the Coronary Artery Disease (CAD) by 2015 in India and of these 23 million would be patients younger than 40 years of age.[1] The four leading causes of death globally in 2030 are projected to be ischemic heart disease, cerebro-vascular disease (stroke), HIV/AIDS, and chronic obstructive pulmonary disease.[2] CAD is usually due to atherosclerosis of large and medium sized arteries, and dyslipidemia has been found to be one of the most significant contributing factors. In Ayurveda, various attempts have been made to use distinctive nomenclature to denote the word dyslipidemia, viz. *Rasagata Sneha Vriddhi*, *Rasa Raktagata Sneha Vriddhi*, *Medodosha*, and *Kapha Medo Margavarana*.

Dyslipidemia can be taken as *Kapha Medo Margavarana*, which denotes *Samprapti* (pathologenesis) rather than a clinical diagnosis. *Asanadi Ghanavati* (AG) [Table 1] is a specific herbal formulation explained in *Shodhanadi Gana Samgraha*."[3] The drugs are indicated specially in the diseases in which *Kapha Dosha* and *Medo Dushya* are vitiated predominantly viz. *Shwitra, Kushtha, Pandu, Prameha, Medoroga*, etc., Most of the drugs have *Ruksha* (dry) *Guna* (property), *Kashaya Rasa* (astringent taste), *Katu Vipaka*, *Lekhana* as well as *Chhedana Karma* and are useful

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in *Kapha Medo Margavarana Janya* condition like dyslipidemia. *Gomutra Haritaki* (GH) is referred for *Kaphaja Shotha Chikitsa*.[4] *Haritaki* (*Terminalia chebula* Retz.) and *Gomutra* (cow urine) are the constituents of this formulation. AG and GH can be helpful to destruction the etiopathogenesis of the disease.

So this study was undertaken to evaluate and compare the clinical effect of AG and GH in patients suffering with the *Kapha Medo Margavarana* (dyslipidemia).

Materials and Methods

Study design

It was a randomized, parallel, and interventional clinical trial. Informed consent was taken from all the patients. Patients (n = 60) with the high lipid profile were selected during October 2011 to April 2012 from Outdoor Patient Department of Kaya Chikitsa, IPGT and RA, Jamnagar and were alternatively divided into two groups (Group A and B). The research protocol was approved by Institutional Ethics Committee (No. PGT/7-A/Ethics/2011-12/2087; dt. 05-09-2011) and registered in CTRI (No.: CTRI/2011/12/002281).

Inclusion criteria

- Patients with age 25 to 60 years
- Elevated levels of serum cholesterol (201 mg/dl or more) and/or elevated serum triglycerides (S. TGs) (151 mg/dl or more) and/or, elevated serum low density lipoprotein (S. LDL) (131 mg/dl or more) and/or elevated serum very low density lipoprotein (S. VLDL) (41 mg/dl or more).[5]

Exclusion criteria

- Patients suffering from type -I DM
- Drug induced dyslipidemia
- Patients with the serious systemic illness such as tuberculosis, carcinoma, and advanced stage of DM.

Investigation

Routine hematological examinations were carried out before and after treatment to rule out any pathological conditions. Bio-chemical test such as complete lipid profile, fasting blood sugar (FBS), serum creatinine, and the blood urea were carried out. Apo-lipoprotein B (Apo-B) as Bio-marker was investigated in 10 patients in each group.

Drugs and posology

Group A

Tablet of AG (500 mg each) was administered in dose of 2 tablets (1 g), thrice a day with *Ushnodaka* (luke warm water) before food for 8 weeks.

Gorup B

Tablet of GH was administered in the same dose and duration of group A.

Follow-up

Follow-up was carried out for 2 weeks after the completion of treatment to see the long-standing effect of the drug.

Pathya-Apathya

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.

Criteria for assessment of the total effect of therapy

The overall effect was assessed on the basis of relief in chief complaints (as subjective criteria) and lipid profile (objective criteria) such as decrease in the serum cholesterol, S. TG, S. LDL, S. VLDL and increase in serum high density lipoprotein (S. HDL).

The overall effect was assessed on following criteria:

- Complete remission – 100% relief
- Markedly improved – <100% to > 75% relief

Table 1: Ingredients of *Asanadi Ghanavati*

Drug name	Latin name	Useful part	Proportion
Asana	*Pterocarpus marsupium* Roxb.	*Sara* (heartwood)	1 part
Tinisha	*Ougeinia dalbergioides* Benth.	*Twaka* (bark)	1 part
Shwetvah-arjuna	*Terminalia arjuna* W. and A.	*Twaka* (bark)	1 part
Prakirya-karanja	*Pongamia glabra* Vent.	*Twaka* (bark)	1 part
Khadira	*Acacia catechu* Willed.	*Sara* (heartwood)	1 part
Kadara	*Acacia suma* Kurz.	*Sara* (heartwood)	1 part
Bhandi-shirisha	*Albizia lebbeck* Benth.	*Panchanga* (entire plant)	1 part
Shishampa	*Dalbergia sissoo* Roxb.	*Sara* (heartwood)	1 part
Meshshrungi	*Gymnema Sylvester* R.Br.	*Patra* (Leaf)	1 part
Shwetachandana	*Santalum album* Linn.	*Kastha* (sapwood)	1 part
Rakta chandana	*Pterocapus santalinus* Linn.f.	*Kastha* (sapwood)	1 part
Daru haridra	*Berberis aristata* DC.	*Kastha* (sapwood)	1 part
Palasha	*Butea frundosa Koen.ex* Roxb.	*Beeja* (seed)	1 part
Aguru	*Aqularia agallocha* Roxb.	*Kastha* (sapwood)	1 part
Shala	*Shorea robusta* Gaertn.f.	*Niryasa* (oleo resin)	1 part
Shaka	*Tectona grandis* Linn.	*Twaka* (bark)	1 part
Kramuka	*Areca catechu* Linn.	*Fala* (fruit)	1 part
Dhava	*Anogeissus latifolia* Wall.	*Twaka* (bark)	1 part
Kalinga	*Holarrena antidysenterica* Wall.	*Bija* (seed)	1 part

- Moderately improved – 75% to > 50% relief
- Mild improvement – 50% to > 25% relief
- Unchanged – 25-0% relief.

Statistical analysis

The Wilcoxon signed rank test method was used to check the significance of the subjective criteria and paired *t* test was used for objective criteria in a single group. To compare the effect of therapy of two groups, Chi-square test was carried out for the subjective criteria and unpaired *t* test for objective criteria. The obtained results were interpreted as, insignificant $P > 0.05$, significant $P < 0.01$, highly significant $P < 0.001$

Observations

Out of 60 patients, the maximum (37%) belonged to age between 45 to 55 years. 50% and 60% patients were male in group A and B respectively. The majority of patients in group A (80%) and group B (70%) were Hindu. 96% of patients of group A and 83.33% of the patients in group B were married. It was observed that, 50% of the females in group A and 40% in group B were housewives in this study. Most of the patients in both groups belonged to middle and rich socio-economic status. In *Nidanas*, *Ahara* was found to be dominant in *Madhura Rasa* (91.33%), *Snigdha Guna* (70%), *Guru Guna* (65%), and *Sheeta Veerya* (63%). *Vishamashana* (68.33%) and *Adhyashana* (36.67%) were found most inappropriate. Maximum patients (90%) were habituated of regular consumption of oily, fried foods, bakery items, curd, etc. In *Vihara*, *Divaswapa* (85%), and *Avyayama* (68.33%) were found most common. In this study, 60% patients had reported of having affection with *Ati-chinta* (stress) and *Krodha* (anger). Most of the patients (71.67%) had gradual onset of disease and the disease was chronic for 1-5 years in 58.33% patients. DM-II and hypertension were observed in 16.67% and 15% patients of both groups. A total of 33.33% patients had a positive family history of dyslipidemia. The majority of patients showed symptoms of *Angagaurava* (76.67%), *Bharavriddhi* (71%), and *Ayase-Shwasa* (53.33%). *Dushti* of *Kapha Dosha*, *Rasa Dhatu*, *Rakta Dhatu*, and *Medo Dhatu* were observed in 78.33%, 88.33%, 30%, and 86.67% respectively. *Dashavidha Pariksha* revealed that 52% of the patients had *Kaphapradhana Pittanubandhi Sharira Prakriti* 67% had *Tamasika Prakriti* 90% were of *Madhyama Sara*, 70% were of *Madhyama Satva*, and 61.67% were of *Madhyama Samhanana*. The study revealed that, 81.67% of the patients had BMI greater than 25 and 70% had body weight between 70 kg and 100 kg.

Results

Effect of therapy on symptoms

In group A, there were relief of symptoms *Angagaurava* (85.37%), *Ayase Swasa* (92.91%), *Bharavriddhi* (6.02%), *Atipipasa* (50%), *Atikshudha* (66.65%), *Daurbalya* (81.81%), *Sandhishool* (57%), *Alasya* (55.09%), and *Sphik-Stana-Udara Avalambana* (47.40%). The relief in *Angagaurava*, *Ayase Swasa*, *Alasya*, *Daurbalya*, *Sphik-Stana-Udara Avalambana* was statistically highly significant ($P < 0.001$). Change of relief on *Sandhishoola* was statistically significant ($P < 0.01$). Statistically insignificant results were found in *Atikshudha*, *Atipipasa* and *Bharavridhhi* ($P > 0.05$) [Figures 1,2].

In group B, relief of symptoms was observed in *Angagaurava* (90.08%), *Ayase Swasa* (87.97%), *Bharavriddhi* (7.05%), *Atipipasa* (75%), *Atikshudha* (73.33%), *Daurbalya* (66.67%), *Sandhishool* (73%), *Alasya* (49.70%), and *Sphik-Stana-Udara Avalambana* (35.61%). The relief in *Angagaurava*, *Ayase Swasa*, *Atipipasa*, *Sandhishool*, and *Sphik-Stana-Udara Avalambana* were statistically highly significant ($P < 0.001$). In *Bharavridhhi*, the relief was statistically significant ($P < 0.01$). Statistically insignificant results were found in *Daurbalya*, *Alasya* and *Atikshudha* ($P > 0.05$) [Figure 1].

Effect on body weight

Body weight was reduced by 2.53% of the subjects in group A, whereas BMI was reduced by 2.59%. In group B, body weight and BMI were reduced by 3.57% and 2.71% respectively. The changes were statistically highly significant ($P < 0.001$) in both the groups [Figure 2].

Effect on body circumference

In group A, circumference of chest, abdomen, hip, pelvis, mid-thigh, and calf were reduced by 0.79%, 1.69%, 1.66%, 0.93%, 2.47%, and 1.09% respectively. In group B, circumference of chest, abdomen, hip, pelvis, mid-thigh, and calf were reduced by 0.45%, 1.12%, 1.80%, 0.93%, 2.34%, and 1.76% respectively. The results were found statistically highly significant ($P < 0.001$) in both the groups [Figure 3].

Effect on skin folds thickness

In group A, skin fold thickness of biceps, triceps, and abdomen was reduced by 5.53%, 5.22%, and 4.64% respectively. In group B, skin fold thickness of biceps, triceps, and abdomen was reduced by 4.99%, 4.84%, and 5.05% respectively. The results were found statistically highly significant ($P < 0.001$) in both the groups [Figure 4].

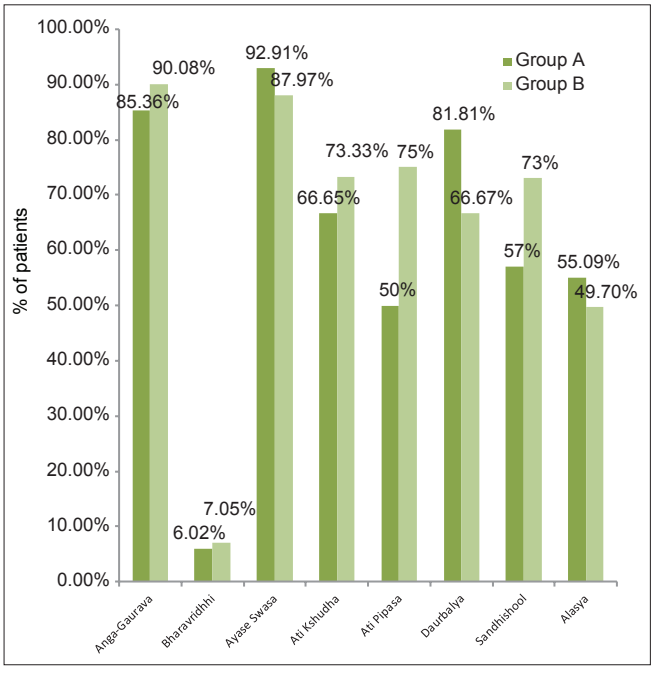

Figure 1: Effect on subjective criteria

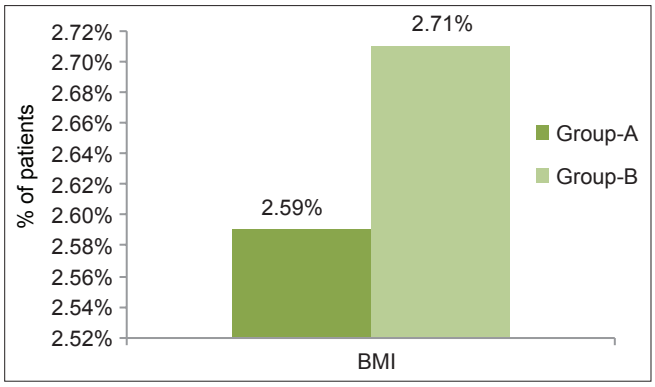

Figure 2: Effect on body mass index

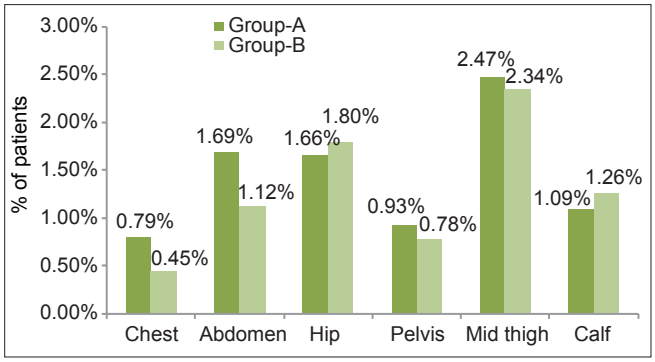

Figure 3: Effect on body circumference

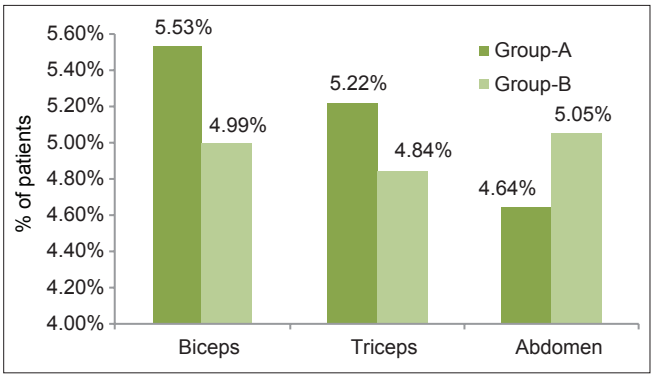

Figure 4: Effect on skin fold thickness

Effect on lipid profile

On statistical evaluation S. cholesterol, S. TGs, S. LDL and S. VLDL were reduced by 7.12%, 7.72%, 11.68%, and 7.73% respectively, whereas S. HDL and apo-lipoprotein were increased by 9.52% and 11.78% respectively in group A. The results were statistically significant in S. cholesterol, S. LDL, S. HDL ($P < 0.01$) and insignificant in S. TG, S. VLDL and apo-lipoprotein ($P > 0.05$) [Table 2].

In group B, S. cholesterol, S. TGs, S. LDL, and S. VLDL were reduced by 6.31%, 9.61%, 12.55%, and 8.99% respectively, whereas S. HDL and apo-lipoprotein were increased by 10.52% and 1.89%. The results were statistically significant in S. cholesterol, S. LDL, S. HDL ($P < 0.01$) and insignificant in S. TGs, S. VLDL and apo-lipoprotein ($P > 0.05$) [Table 3].

Effect on bio-chemical parameters

In group A, 9.65% reduction was observed in FBS level, 5.15% decrease in S. creatinine, 0.97% increase in S. urea which is Within Normal Limit (WNL), although in group B 0.22% increase in FBS level (which is WNL), 1.0% decrease in S. creatinine, and 10.91% decrease in S. urea.

Comparison between both the groups

On comparison of both Groups, the un-paired *t* test showed insignificant difference in the objective parameters [Table 4]. While comparing the effect of therapy over the subjective criteria by using Chi-square analysis, both the groups showed similar effect as the results were insignificant [Table 5].

Overall effect of therapy

In group A, 27 patients had completed the course of treatment. Out of them moderate improvement was observed in 4 (14.81%) patients, mild improvement was observed in 19 (70.37%) patients, whereas no improvement was observed in 4 (14.81%) patients.

In group B, 27 patients had completed the course of treatment; out of them moderate improvement was observed in 1 (3.70%) patient, mild improvement was observed in 20 (74.07%) patients, whereas no improvement was observed in 6 (22.22%) patients.

Discussion

The term *Margavarana* signifies the obstruction in the channels; in this study *Margavarana* refers to obstruction in *Raktamarga* rather than other channels within the body. S. cholesterol in patients of groups A and B were decreased by 7.12% and 6.31% respectively, which was statistically significant ($P < 0.05$). S. cholesterol is the total level of cholesterol that is found in the blood stream. Moreover, it helps to have a rough idea regarding the balance between the HDL and LDL cholesterol. Hence, the reduction in cholesterol levels that is significant has to be considered as a positive outcome of the trial drugs. On S. TG both drugs showed a decrease by 7.72% and 9.29% respectively, which was statistically insignificant. When absorption of triglyceride in the intestine is proper, it may reduce S. TG level. Over production of VLDL and triglyceride in the liver has been proposed to be driven by high levels of Serum free fatty acid in patients with insulin resistance.[6] The ingredients of AG and GH are hepato-protective and anti-hypercholesterolemic. Hence, it can be understood that the action of drug is mainly on the endogenous/hepatic pathway of lipids. Both drugs showed S. VLDL decrease by 7.73% and 8.99% with a mean difference of 16.54 and 11.76 respectively, but it was statistically insignificant. VLDL contains the highest amount of TGs, and it is also considered as bad cholesterol as it helps cholesterol build up on walls of arteries. Hence, drop in the mean VLDL value attained in working groups is supporting the lipid lowering efficacy of AG and GH.

On S. LDL both drugs showed a decrease by 11.68% and 12.55% respectively, which was statistically significant. In the present study, mean cholesterol level was decreased, and mean HDL level was increased, which directly indicates the reduction of LDL level. The probable mechanism may be that the trial drugs reduce intestinal absorption of cholesterol by reducing

Table 2: Effect of therapy on objective criteria in Group A

Parameter (mg/dl)	Mean score		Difference	%	Paired t test				Significance
	BT	AT			SD	SEM	*T*	*P*	
Serum cholesterol	222.04	206.22	15.81	7.12↓	27.93	5.38	2.941	0.007	S
S. TG	261.15	241.00	20.15	7.72↓	82.65	15.91	1.267	0.217	NS
S. LDL	127.77	112.82	14.93	11.68↓	25.70	4.95	3.018	0.006	S
S. VLDL	52.24	48.20	4.385	7.73↓	16.54	3.18	1.268	0.216	NS
S. HDL	41.29	45.22	3.93	9.52↑	7.36	1.416	–2.772	0.01	S
Apo-B	99.75	111.50	–11.75	11.78↑	26.91	9.51	–1.235	0.257	NS

S. LDL: Serum low density lipoprotein; S.VLDL: Serum very low density lipoprotein; S. HDL: Serum high density lipoprotein; Apo-B: Apo-lipoprotein B; S.TG: Serum triglyceride; AT: After treatment; BT: Before treatment; SD: Standard deviation; SEM: Standard error of mean; S: Significant; NS: Non-significant

Table 3: Effect of therapy on objective criteria in Group B

Parameter (mg/dl)	Mean score		Difference	%	Paired *t* test				Significance
	BT	AT			SD	SEM	*T*	*P*	
Serum cholesterol	212.44	199.04	13.40	6.31↓	33.52	6.45	2.078	0.048	S
S. TG	244.30	221.59	22.70	9.29↓	57.94	11.15	2.036	0.052	NS
S. LDL	123.39	107.90	15.49	12.55↓	28.96	5.57	2.781	0.01	S
S. VLDL	48.70	44.32	4.38	8.99↓	11.76	2.26	1.938	0.064	NS
S. HDL	40.11	44.33	4.22	10.52↑	6.880	1.324	–3.189	0.004	S
Apo-B	103.73	105.73	–2.00	1.89↑	20.40	6.15	–0.325	0.752	NS

S. LDL: Serum low density lipoprotein; S.VLDL: Serum very low density lipoprotein; S. HDL: Serum high density lipoprotein; Apo-B: Apo-lipoprotein B; S.TG: Serum triglyceride; AT: After treatment; BT: Before treatment; SD: Standard deviation; SEM: Standard error of mean; S: Significant; NS: Non-significant

Table 4: Comparison between both the groups (subjective parameters)

Subjective parameter	Group	≤50%	>50%	Chi square value	'*P*' value	Significance
Anga-gaurava	A	02	15	0.03	0.87	NS
	B	02	22			
Bhara-vriddhi	A	24	01	0.444	0.505	NS
	B	26	01			
Ayase- shwas	A	01	10	0.00	0.98	NS
	B	03	15			
Ati-Kshudha	A	01	02	0.18	0.68	NS
	B	01	04			
Ati-Pipaasa	A	02	01	0.03	0.85	NS
	B	03	05			
Daurbalya	A	02	08	0.938	0.333	NS
	B	03	02			
Sandhishool	A	05	02	1.371	0.24	NS
	B	01	04			
Alasya	A	03	09	3.403	0.06	NS
	B	05	01			

NS: Non-significant

the LDL particle concentrations. In AG; *Asana* (*Pterocarpus marsupium* Roxb.), *Arjuna* (*Terminalia arjuna* W. and A.), *Shirisha*, *Rakta-Chandana* (*Pterocapus santalinus* Linn.f.), *Shaka* (*Tectona grandis* Linn.); and in GH, both *Gomutra*[7] and *Haritaki*[8] may have anti-oxidant activity. Both drugs may reduce oxidative stress by preventing lipid peroxidation with more than two double bonds (free radicals). In both groups, S. HDL was increased by 9.52% and 10.52% respectively, which was statistically significant. HDL is considered as good cholesterol because of its effectiveness in cholesterol removal from the periphery to liver (reverse cholesterol transport). Drugs probably inhibit an enzyme in the liver that is involved in triglyceride synthesis, causing a decrease in VLDL production. It in turn leads to reduction in free fatty acids in the visceral fat levels, and thereby improvement in the HDL levels.

Apo-B 100 is the primary apo-lipoprotein of the LDL which is responsible for carrying cholesterol to the tissues. The normal level is considered as 40-125 mg/dl. In the present study, both groups showed an increase in Apo-B level but were within normal limits. In AG group, FBS was reduced by 9.55% although in GH group, FBS was increased by 0.22%, but it is within normal limit.

Table 5: Comparison between both the groups (objective parameters)

Parameters	Group	*n*	Difference in means	Unpaired '*t*' test				
				S.D.	S.E.M	'*t*'	'*P*'	Significance
Serum cholesterol	A	27	15.815	27.938	5.377	0.287	0.776	NS
	B	27	13.407	33.522	6.451			
Serum triglyceride	A	27	20.148	82.655	15.907	–0132	0.896	NS
	B	27	22.704	57.938	11.150			
S. LDL	A	27	14.933	25.709	4.948	–0.075	0.940	NS
	B	27	15.496	28.956	5.573			
S.VLDL	A	27	4.037	16.537	3.183	–0.089	0.929	NS
	B	27	4.385	11.756	2.262			
S.HDL	A	27	–3.926	7.359	1.416	0.153	0.879	NS
	B	27	–4.222	6.880	1.324			
Apo-lipoprotein B	A	08	–11.75	26.906	9.513	–0.901	0.380	NS
	B	11	–2.000	20.396	6.150			

S. LDL: Serum low density lipoprotein; S.VLDL: Serum very low density lipoprotein; S. HDL: Serum high density lipoprotein; Apo-B: Apo-lipoprotein B; S.TG: Serum triglyceride; SD: Standard deviation; SEM: Standard error of mean; S: Significant; NS: Non-significant

Ingredients of AG such as *Asana*, *Karanja* (*Pongamia glabra* Vent), *Khadira* (*Acacia catechu* Willed), *Mesha-sringi* (*Gymnema Sylvester* R.Br), *Daruharidra* (*Berberis aristata* DC.), *Shaka*, and *Kalinga* are established hypoglycemic drugs. This study proves that, AG is an ideal drug which can lower the BSL (in pre-diabetics and diabetics).

AG and GH both showed improvement on *Angagaurava*, which was decreased by 85.36% and 90.08% respectively. *Guru Guna*, which is mainly responsible for *Angagaurava* is also a causative factor for increasing *Ama* resulting in formation of *Pralepa* in *Rasavaha* and *Raktavaha Srotasa*. *Tikshna*, *Ushna*, and *Ruksha Guna* of GH help to subside *Gaurava* by clearing *Sroto-avarodha*. AG showed improvement on *Daurbalya*, which was decreased by 81.81%, which is mainly due to *Sroto-Avarodha*, which ultimately hampers the nutrition of next *Dhatu*. AG decreases *Kleda* as well as *Sroto-Avarodha*. *Atikshudha* and *Atipipasa* are due to *Tikshnata* of *Pachakagni* and increased *Vata* in *Koshtha Sthana*.[9] GH increase *Jatharagni* by *Amapachana*. It clears *Sroto-avarodha* and *Medomargavarana*. Ultimately, *Prakruta Gati* of *Vata* leads to *Samyaka Kshudha* and *Pipasa*. *Sandhishoola* is due to *Bharavridhhi* (*Rasagata Sama Shleshma*) or *Samata* (*Medogata Sama Sleshma*). *Amapachana* property of GH decreases *Sandhishoola* by converting *Sama* condition into *Nirama*. *Tikta*, *Kashaya*, *Sheeta*, *Pitta-Kaphahara* property of AG decrease *Kleda* and *Meda* as well as it reduces *Swedadhikya* and *Daurgandhya*. *Alasya* is due to *Hetu* like *Guru*, *Snigdha*, *Madhura Ahara*, which produce *Ama*, *Kleda*, *Abhishyanda*, *Sroto-Avarodha*. AG has *Laghu*, *Ruksha* and *Tikta-Kashaya* property. It helps in *Kleda Upashoshana*, decreases *Abhishyanda*, *Sroto-Avarodha* and *Alasya*. *Ayase Swasa* is one of the symptoms of *Medo Vridhhi*. Reduction in *Apachita Medovriddhi* (*Medakshaya*) showed improvement on the symptoms like *Ayase Swasa*. *Nidradhikyata* (*Tamo Sleshma Samudbhava*) is either due to *Sroto-avarodha* or *Manasa Tamo Adhikya* (*Manovaha Srotasa*).

Probable mode of action of AG

Due to *Kleda Dustikara Nidana*, it increases *Kleda*, *Sleshma*, and *Snehamsha* in *Rasa*, *Rakta*, and *Medo Dhatu*. *Guru* and *Snigdha Guna* manifest symptoms such as *Medovridhhi*, *Sphik-Stana-Udara Avalambana*, *Ayase Swasa*, *Daurbalya*, *Daurgandhya*, *Swedadhikya* result in *Kleda Bahula Samprapti Janya Vyadhi* such as *Prameha*, *Kustha*, and *Meda-roga*. AG has *Laghu*, *Ruksha*, *Sheeta*, *Tikta*, *Kashaya*, and *Katu Vipaka*. *Tikta*, *Kashaya* and *Sheeta* having action of *Kleda*, *Medo*, *Vasa*, *Mutra*, *Sweda Shoshana*, *Shodhana* and *Rakta Prasadana*, which decrease *Raktagata Kleda* and *Meda*. Ultimately reduce *Medoshaithilya*. AG probably works at the level of *Medodhatwagni*. Ingredients of AG, *Asana*[10] having action of anti-hyperlipidemic, anti-diabetic, cardiac tonic; *Tinisha* (*Ougeinia dalbergioldes* Benth.)[11] having anti-inflammatory, cancer preventive, hypocholesterolemic; *Arjuna*[12] having cardiotonic, antihyperlipidemic, antiatherogenic, antioxidant; *Karanja*[13] having hypolipidemic, hypoglycemic action. It was observed that methanolic bark extract of *Kadara* (*Acacia suma* Kurz.)[14] is potential to prevent the secondary complications of diabetes mellitus like atherosclerosis. *Daruharidra*[15] has hypoglycemic, hypolipidemic, and anti-inflammatory activities; *Dhava* (*Anogeissus latifolia* Wall.)[16] shows hypolipidemic and hepatoprotective activity, whereas *Khadira*[17] is well-known of having anti-dyslipidemic and anti-diabetic property.

Anti-inflammatory drugs are potential enough for endothelial protection by reducing endothelial injury. Anti-oxidant drugs reduce chances of atherosclerotic complications like CVA (cerebrovascular accident), whereas hypoglycemic drugs improve carbohydrate metabolism and glycogenesis by reducing excess lipogenesis. Hepatoprotective drugs enhance the enzyme useful for HDL production. Over all AG is cardioprotective and hepatoprotective. Ultimately, it is useful to regulate lipid metabolism

Probable mode of action of GH

Due to *Dustikara Nidana*, it increases *Sama Sleshma*, *Agnimandhya* and *Amarasa*. *Rasagata Ama* manifests symptoms such as *Apachita Medo Dhatu Vridhhi*, *Bharavridhhi*, *Angagaurava*, *Sroto-avarodha*. And *Sama Sleshma* causes symptoms such as *Angagaurava*, *Sroto-avarodha*, *Ati Kshudha*, *Ati Pipasa*, and *Asthi-Sandhishoola*. It may lead to *Ama Bahula Samprapti Janya Vyadhi* such as *Medoroga*, *Madhyama Margavaranajanya Vyadhi*, *Trimarmiya Vyadhi*. *Gomutra* (of

GH) possesses *Katu Rasa* and *Ushna Virya*, whereas *Haritaki* has property of *Doshanuloman*, *Dipana-pachana*, *Sroto Vibandhanahara*, *Pramehahara*, *Hridaya-urah Pralepahara*, and *Budhhi Indriya Bala Prada*. GH increases *Jatharagni* by *Amapachana* reducing *Sama Sleshma* and *Apachita Medo Dhatu*. GH probably works at the level of *Jatharagni*.

Triglyceride and other cholesterol are usually absorbed in gastro-intestinal tract (GIT). When *Jatharagni Vikriti* occurs, it manifests in *Ama*, disturbs lipid metabolism, increases triglyceride and other cholesterol. GH improves physiological digestion process at the level of the intestine and liver. It also regulates lipid metabolism. *Gomutra*[18] (bio-enhancer) can enhance the efficacy and effect of other drugs. *Haritaki*[19] has anti-hyperlipidemic, anti-oxidant, cytoprotective, anti-diabetic, cardioprotective as well as hepatoprotective activity. Over all, GH is cardioprotective and hepatoprotective, and it promotes physiological digestive process.

Conclusion

On comparison, both drugs *Asanadi Ghanavati* and *Gomutra Haritaki* showed better result on dyslipidemia. *Asanadi Ghanavati* is more useful in pre-diabetic and diabetic, dyslipidemic patients while *Gomutra Haritaki* has an additional advantage of improving physiological digestive process. *Asanadi Ghanavati* probably works at the level of *Medodhatwagni*. *Gomutra Haritaki* probably works at the level of *Jatharagni*. *Asanadi Ghanavati* and *Gomutra Haritaki* should be used in *Kleda Bahul Samprapti Janya Vyadhi* and *Ama Bahul Samprapti Janya Vyadhi* respectively. The ingredients of *Gomutra Haritaki* are cost-effective and easily available throughout the country. Finally, *Gomutra Haritaki* is a potent drug in the management of dyslipidemia both as a prophylactic and curative agent.

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हिन्दी सारांश

डिस्लेपिडेमिया मे असनादि धनवटी और गोमूत्र हरितकी का चिकित्सकीय तुलनात्मक अध्ययन

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डिस्लेपिडेमिया, लिपोप्रोटीन के अधिक उत्पादन या कम उत्पादन के कारण होनेवाली चयापचय प्रक्रिया की व्याधि है । उसे आयुर्वेद में कफमेदोमार्गावरण, अति स्थौल्य या प्रमेह के साथ तुलना कर सकते है । असनादि घनवटी, अष्टांग हृदय मे वर्णित असनादि गण का परिवर्तित स्वरुप है और गोमूत्र हरितकी चरक संहिता मे शोथ चिकित्सा एवं अष्टांग हृदय अर्श चिकित्सा मे वर्णित है । इस वर्तमान अध्ययन का उद्देश्य समस्त जन समुदाय के लिये सुरक्षित एवं किफायती शमन चिकित्सा खोजना है । इस अध्ययन के लिये हाई लिपीड प्रोफाईल वाले रुग्ण को ओ.पी.डी. काय चिकित्सा, आई.पी.जी.टी.एण्ड आर.ए., जामनगर से चयन किया गया है और अनियमित रुप से दो वर्ग मे विभाजित किया गया । इस यादृच्छिक, इन्टरवेन्शनल, समान्तर परिक्षण मे असनादि घनवटी और गोमूत्र हरितकी १ ग्राम दिन मे ३ बार, ८ सप्ताह के लिये, ६० रुग्णों को दिया गया । जिनमे प्रत्येक वर्ग मे २७ रुग्णों ने चिकित्सा क्रम पूर्ण किया । अध्ययन के परिणाम से पता चला है कि, असनादि घनवटी से सीरम कोलेस्टेरोल ७.१२%, सीरम ट्रायग्लिसेराईड ७.७२%, सीरम एल.डी.एल. ११.६९%, सीरम वी.एल.डी.एल. ७.७३% कम हुआ एवं सीरम एच.डी.एल. मे ९.५२% वृद्धि हुई । जिसमे १४.८१% रुग्ण मे मध्यम सुधार एवं ७०.३७% रुग्ण मे अल्प सुधार पाया गया । गोमूत्र हरीतकी से सीरम कोलेस्टेरोल ६.३१%, सीरम ट्रायग्लिसेराईड ९.२९%, सीरम एल.डी.एल. १२.५५%, ८.९९% कम हुआ एवं सीरम एच.डी.एल. मे १०.५२% कम हुआ एवं सीरम एच.डी.एल. मे १०.५२% वृद्धि हुई । जिसमे ३.७० रुग्ण मे मध्यम सुधार एवं ७४.०७% रुग्ण मे अल्प सुधार पाया गया । इस अध्ययन के अनुसार असनादि घनवटी एवं गोमूत्र हरितकी का क्रमशः क्लेदबहुल संप्राप्तिजन्य व्याधि एवं आमबहुल संप्राप्तिजन्य व्याधि में प्रयोग करने का सुझाव दिया जा सकता है ।