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Comparative evaluation of efficacy of *Kulattha Gutika* with Atorvastatin in the management of Dyslipidemia (*Medoroga*) - RCT

Research Article

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Abstract

Introduction: Dyslipidemia is the disorder of lipoprotein metabolism manifested by an elevation of the total cholesterol, the low-density lipoprotein (LDL) cholesterol, the triglyceride concentrations, and a decrease in the high-density lipoprotein (HDL) cholesterol concentration in the blood. Today's unhealthy food habits and sedentary lifestyle are the main causative factors for Dyslipidemia. It is not defined in Ayurveda, it can be related to a number of conditions, including *"Medoroga, Rasa Raktagata Snehavruddhi Shonitabhishyandana,* and *Dhamanipratichaya"* based on similarities in etiology, pathogenesis, and manifestations. Aim: Comparative evaluation of Efficacy of *Kulattha Gutika* and Atorvastatin in the Management of Dyslipidemia (*Medoroga*). Material and Methods: Study comprises total 60 patients of Dyslipidemia randomly divided into two equal groups. Group A (Experimental group) was treated with 1gm *Kulattha Gutika* thrice a day before meals with warm water and Group B (Control group) was treated with 10 mg Atorvastatin at night before sleep with water for 45 days. Patients were assessed for Objective parameters like Lipid profile (S. TCH, TG, LDL, HDL, VLDL) and BMI was done on 0 and 45th day. Result: Statistically significant improvement was observed in objective parameters like TCH, HDL, LDL, VLDL, and TG in both groups. But in BMI Statistically significant improvement was observed in group A, treated with *Kulattha Gutika*. Conclusion: *Kulattha Gutika* is as efficacious as Atorvastatin in managing Dyslipidemia (*Medoroga*) and is an economical alternative without adverse effects.

Keywords: Dyslipidemia*, Medoroga, Kulattha Gutika,* Atorvastatin, Lipid profile.

Introduction

Dyslipidemia is a very common metabolic disorder due to irregular dietary habits, quality of food, lack of physical exercise added to a stressful lifestyle, and other factors that lead to higher or fluctuating levels of free fatty acids. All these are collectively responsible for altering the body's metabolic activities, and these factors lead to changes in lipid profiles.

A condition of disrupted lipid metabolism that involves abnormalities in one or more of the lipoproteins present in the blood is called Dyslipidemia (1). Total cholesterol (TCH), low-density lipoprotein (LDL) cholesterol, triglyceride concentration (TG), and a reduction in the high-density lipoprotein (HDL) cholesterol concentration in the blood are manifestations of Dyslipidemia (2). The prevalence of hyper-cholesterolemia was 13.9 percent, hyper-triglyceridemia was 29.5%, low S.HDL-C was 72.3%, and high S. LDL-C levels were 11.8%, according to the ICMR-INDIAB study (3). In Dyslipidemia, the level of circulating lipids or the lipoprotein fraction is abnormal due to genetic or environmental factors that induce catabolism to occur after plasma lipoprotein is produced and cleared from circulation (4). The NCEP ATP III guidelines describe hyperlipidemia as having total cholesterol (TC) > 200 mg/dl, S.LDL cholesterol > 130 mg/dl, S. triglyceridemia (TG) > 150 mg/dl, and S. HDL-C (HDL cholesterol) < 40 mg/dl (5). Hyper-cholesterolemia, specifically a raised plasma level of cholesterol carried in LDL, is Dyslipidemia that is most clearly related to an increased risk of CAD (coronary artery disease)(6).

As per Ayurveda, Dyslipidemia can be correlated with *Medo-Dushti* (vitiation of fat tissue) in *Rasa* (primary product of digested food) or *Rakta Dhatu* (blood tissue). Digestive fire is responsible for all the metabolic activities in the human body. Excessive use of heavy and unctuous food items, overeating, anxiety, stress, lack of physical exercise, habitual excessive alcohol intake, and a sedentary lifestyle cause vitiation of digestive factors leading to *Rasavaha* and *Medovaha Strotodushti* (vitiation of channels carrying nutrient fluids and fat tissue(7,8). Due to the weakness of metabolic factors of the body, the food that is not completely digested yields an immature primary product of digested food in the stomach, and due to the retention, it undergoes fermentation. This primary

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product of partially digested food is called *Ama* (9). This can be produced at the level of *Jatharagni* (metabolic factors located in the digestive tract)) or *Dhatwagni* (metabolic factors located at the tissue level). Improper function of *Agni* (metabolic factors of the body) is stated to be the cause of *Ama* (partially metabolized product of digested food) production(10). This *Aam* produced at this level inhibits the assimilation of nourishing factors leading to an abnormal increase of circulating *Snehansha* (unctuousness) resulting in the excessive formation and accumulation of *Abaddha Meda Dhatu* (fat tissue) which leads to *Medoroga*. It causes symptoms like increased fatigue on exertion, dyspnea, and accumulation of fat at various sites in the body (11,12). The basic line of treatment includes *Nidanparivarjana* (avoidance of etiological factors). *Chikitsa* mainly includes *Apatarpana* (depletion therapy), *Shodhana* (purification therapy), and *Shamana* (palliative therapy) (13,14).

Shamana Chikitsa includes the use of drugs having *Laghu*, *Ruksha*, *Ushna*, *Tikshna*, *Vata-Kaphanashak*, *Medohar*, and *Lekhana* properties. *Kulattha* possesses all these properties and is mentioned in *Bhavprakash* (15) for managing *Medoroga*. Hence it may help in *Samprapti Vighatana* (breaking pathogenesis). Atorvastatin is a lipid-lowering drug of the statin group used as a standard drug in this study.

Need of study

Dyslipidemia is a commonly encountered lifestyle disorder in day-to-day practice. It is prevalent in both sexes and mostly in advanced age. Dyslipidemia is considered a major risk factor that leads to cardiovascular and cerebrovascular diseases. Numerous research studies conducted on lipid disorders, but no any research proved complete remission therapy of it. In modern medicine, various classes of drugs like HMG-CoA reductase inhibitors- statins, Bile acid-binding resins, Nicotinic acid (niacin), Fibric acid derivatives, Cholesterol absorption inhibitors (ezetimibe) are used but long-term use of them are associated with adverse effects like myalgia, sleep disturbances, and risk of liver or kidney damage(16,17). *Shodhana Chikitsa* is effective in lowering lipid levels but cannot be used in patients having contraindications for it. It is relatively expensive and needs repeated hospital visits. In these conditions, *Shamana Chikitsa* is the best choice. In *Shamana Chikitsa*, *Guggul* is the primary ingredient in the majority of the formulations. Use of *Guggul* for a longer period can cause side effects like impotency (18) and gastrointestinal discomfort (19) as per *Bhavaprakash Samhita*. Some patients may have gastric irritation from the *Lekhaniya Mahakashaya* medications that are available to treat Dyslipidemia. *Kulattha is* included in *Anna Varga*. It is cost-effective easily available. The antihyperlipidemic action of *Kulattha* was demonstrated in animal experiments(20), but no human trials have been done. Therefore, the purpose of the current study was to assess the effectiveness of *Kulattha* in treating Dyslipidemia in humans.

Aim

Comparative evaluation of Efficacy of *Kulattha Gutika* and Atorvastatin in the Management of Dyslipidemia (*Medoroga*).

Objectives

- To assess the efficacy of *Kulattha Gutika* on Lipid profile (S. TCH, TG, LDL, HDL, VLDL) and BMI.
- To assess the efficacy of Atorvastatin on Lipid profile (S. TCH, TG, LDL, HDL, VLDL) and BMI.
- To compare the efficacy of *Kulattha Gutika* and Atorvastatin on Lipid profile (S. TCH, TG, LDL, HDL, VLDL) and BMI.
- To study the incidence of Dyslipidemia as per *Prakruti.*

Material and Methods

Source of Data

Patients visited the outdoor and indoor *Kayachikitsa* department and from the specialty, camps were registered for the present study. After institutional ethics committee approval (Ref no. MGACHRC/IEC/ July-2021/339) or CTRI registration. (CTRI/ 2021/12/038818).

Study design: Randomized single-blind (Accessor) Standard Controlled equivalence Trial
Study Type: Interventional Study.
Sampling procedure: Lottery method (divided 30 patients in each group)
Interventions: *Kulattha Gutika*
Sample Size: 60 (30*2)

Inclusion criteria

- Patients, willing to participate in the study
- Age ranges from 30 to 60 years of both genders.
- Patients having the following lipid ranges-
- [ATP-III National Cholesterol Education Program (NCEP) criteria](21)
    - Serum Total Cholesterol ≥200 mg/dl & or
    - Serum Triglycerides = 150-499 mg/dl & or
    - Serum HDL Cholesterol< 40 mg/dl & or
    - Serum LDL Cholesterol = 130 -189 mg/dl

Exclusion criteria

- Already diagnosed patients having illnesses such as CVD (cardiovascular disorders), DM (Diabetes mellitus type II), and kidney diseases.
- Patients on medicines such as corticosteroids.
- During pregnancy as well as lactation period in women.

Composition of material

Table 02: Ingredients of *Kulattha Gutika*(23)

Ingredient	Latin Name	Used Part	Quantity
Kulattha	*Dolichos Biflorus Linn.*	Grain	1 part

Posology: (As per PICO model)

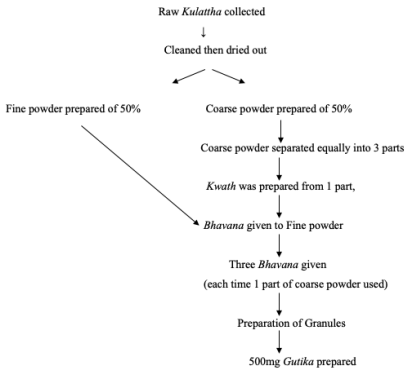
Table 01: Showing Posology of drugs

Group	Sample size	Intervention	Dose and frequency	Anupan	Duration	Follow-up
A	30	*Kulattha Gutika*	1gm(22) Three times per day (before food)	Luke warm water	45 days	15th,30th& 45th day during treatment
B	30	Tab. Atorvastatin	10 mg at night			

Preparation of Material

Kulattha Gutika was prepared in Dattatreya *Rasashala* of our Institute as per the standard operating procedure mentioned in *Sharangdhara Samhita*(24).

Flow chart 01: Showing preparation of *Gutika*



Investigation: FBS, Lipid profile **(**TCH, TG, HDL, LDL, and VLDL**)**

Assessment Criteria

- Lipid profile (before & after treatment) (TCH, TG, HDL, LDL, and VLDL)
- Body Mass Index (before & after treatment)
- To assess the *Prakruti* as per the AYU SOFT software application(25)

Statistical analysis

The software used in the study was SPSS 27.0 version and Graph Pad Prism 7.0 version, and $p < 0.05$ is considered to be the significant level. Descriptive and inferential statistics were applied in the statistical analysis, using the Chi-square test, Student's paired and unpaired t-test.

Observation and Results

Table 03: Distribution of patients according to Demographic characteristics

Demographic characteristics	Group A (n=30)	Group B (n=30)	χ2-value/ t-value	p-value
Average age in years	42±9.04	44.10±7.91	0.91	0.34, NS
Age Range	30-60 yrs	30-60 yrs		
Gender				
Male	16(53.33%)	14(46.67%)	0.26	0.70, NS
Female	14(46.67%)	16(53.33%)		
Occupation				
Service	19(63.33%)	8(26.67%)	4.05	0.44, NS
Business	1(3.33%)	6(20%)		
Labourer	7(23.33%)	7(23.33%)		
Housewife	3(10%)	9(30%)		
Socio-economic Status				
Poor	6(20%)	11(36.67%)	2.05	0.15, NS
Middle	24(80%)	19(63.33%)		
Prakruti				
Kapha Pittaj	6(20%)	10(33.33%)	4.77	0.44, NS
Kapha Vataj	13(43.33%)	11(36.67%)		
Pitta Kaphaj	1(3.33%)	0(0%)		
Pitta Vataj	0(0%)	2(6.67%)		
Vata Kaphaj	5(16.67%)	3(10%)		
Vata Pittaj	5(16.67%)	4(13.33%)		
Agni				
Manda	20(66.67%)	19(63.33%)	0.07	0.78, NS
Vishama	10(33.33%)	11(36.67%)		
Family History				
Yes	6(20%)	5(16.67%)	0.11	0.73, NS
No	24(80%)	25(83.33%)		
Diet				
Vegetarian	11(36.67%)	10(33.33%)	0.07	0.78, NS
Mixed	19(63.33%)	20(66.67%)		

Fry Food				
Yes	23(76.67%)	23(76.67%)	-	-
No	7(23.33%)	7(23.33%)		
Diwa Swapna				
Yes	17(56.67%)	11(36.67%)	2.41	0.12, NS
No	13(43.33%)	19(63.33%)		
Habit				
Not Any	11(36.67%)	9(30%)	3.70	0.44, NS
Alcohol	2(6.67%)	1(3.33%)		
Smoking	4(13.33%)	4(13.33%)		
Tea	4(13.33%)	10(33.33%)		
Tobacco	9(30%)	6(20%)		
Exercise				
Yes	8(26.67%)	15(50%)	3.45	0.06, NS
No	22(73.33%)	15(50%)		

Demographic data

In this study, the incidence of Dyslipidemia was observed more in the fifth decade of life and equal in both sexes in both groups. The prevalence of Dyslipidemia was more in patients engaged in service as well as housewives, the maximum number of patients (71.66%) belonged to the middle socio-economic class, and family history was absent in the maximum number of patients (81.66%). The maximum number of patients had mixed types of dietary habits(65%) and fried food (76.67%), the majority (64%) of patients had *Mandaagni,* and the history of *Diwaswapna* was present in 56% of patients.

The majority of patients in this study (73.33%) were not practicing *Vyayam* (physical exercise), one-third of patients had no addiction, and the remaining were addicted to alcohol, smoking, tobacco chewing, and tea.

In this study, the majority of patients had *Kaphavataja* followed by *Kaphapittaja Prakruti*. Thus, *Kapha* predominantly either associated with *Vata* or *Pitta*, is more in predisposing to the disease *Medoroga* and Dyslipidemia(26). From this it can be stated that there is a strong association between *Kaphaja Prakriti* and the incidence of Dyslipidemia.

Table 04: Comparison of Effect of therapy on BMI in both groups before and after treatment

Group	BT	AT	Student's Paired "t test & t-value"	p-value
A	25.48±0.93	25.23±0.81	7.24	0.0001, S
B	25.51±1.60	25.53±1.61	0.85	0.40, NS
Comparison of both groups ("Student's unpaired t test")				
t-value	0.06	0.89		
p-value	0.94, NS	0.37, NS		

Effect on BMI

In this study, the mean score of BMI before treatment was 25.48±0.93 and 25.51±1.60 which was reduced to 25.23±0.81 and 25.53±1.63 in group A and group B respectively after treatment. BMI showed statistically significant improvement after treatment in group A with a p-value of 0.0001 and group B had statistically non-significant improvement after treatment with P-value0.40 as shown in table no 04. *Kulattha* has *Kaphavata Nashaka* and *Lekhana* properties which cause scraping action removing excessive accumulated *Meda* and *Kapha* thus causing a reduction in body weight and BMI. Comparison of both groups in reduction of BMI after treatment was statistically non-significant (p- 0.37) showing equal effects of both groups on BMI the cause may be due to the short time duration of the present trial.

Table 05: Comparison of Effect of therapy on TCH in both groups before and after treatment

Group	**BT**	**AT**	**Student's Paired "t-test & t-value"**	**p-value**
A	213.23±46.32	195.60±42.24	4.97	0.0001, S
B	234.63±31.05	209.16±27.64	9.27	0.0001, S
Comparison of both groups ("Student's unpaired t test")				
t-value	2.10	1.47		
p-value	0.14, NS	0.14, NS		

Table 06: Comparison of Effect of therapy on HDL in both groups before and after treatment

Group	BT	AT	Student's Paired "t-test& t-value"	p-value
A	37.36±9.84	40.63±8.59	3.15	0.004, S
B	42.73±10.52	45.40±7.75	2.30	0.029, S
Comparison of both groups ("Student's unpaired t test")				
t-value	2.01	2.25		
p-value	0.46, NS	0.028, S		

Table 07: Comparison of Effect of therapy on LDL in both groups before and after treatment

Group	BT	AT	Student's Paired "t-test& t-value"	p-value
A	131.60±47.98	117.90±42.05	3.46	0.002, S
B	146.46±28.60	125.13±28.81	7.89	0.0001, S
Comparison of both groups ("Student's unpaired t test")				
t-value	1.45	0.77		
p-value	0.15, NS	0.44, NS		

Table 08: Comparison of Effect of therapy on VLDL in both groups before and after treatment

Group	BT	AT	Student's Paired "t-test& t-value"	p-value
A	44.26±13.96	37.53±12.07	6.56	0.0001, S
B	44.46±15.26	38.30±12.50	5.32	0.0001, S
Comparison of both groups ("Student's unpaired t test")				
t-value	0.05	0.24		
p-value	0.95, NS	0.81, NS		

Table 09: Comparison of Effect of therapy on TG in both groups before and after treatment

Group	BT	AT	Student's Paired "t-test& t-value"	p-value
A	222.80±68.98	187.53±60.59	6.79	0.0001, S
B	221.80±76.50	189.70±61.95	5.49	0.0001, S
Comparison of both groups ("Student's unpaired t test")				
t-value	0.05	0.13		
p-value	0.95, NS	0.89, NS		

Effect on Lipid Parameters

In this study, mean score of TCH before treatment was 213.23±46.32and234.63±31.05 which was reduced to 195.60±42.24 and 209.16±27.64 in group A and group B respectively after treatment. TCH showed statistically significant improvement after treatment in both groups with a p-value of 0.0001.

Comparing the two groups was done using the Student's Unpaired t-test, and the results were statistically non-significant before and after completion of treatment with p-value 0.14 and 0.14 respectively, that is both trial and study groups are similar at baseline and have equal efficacy in reducing TCH as shown in table no 05.

In this study, mean score of HDL before treatment was 37.36±9.84 and 42.73±10.52 which was increased to 40.63±8.59and 45.40±7.75in group A and group B respectively after treatment. HDL showed statistically significant improvement after treatment in both groups with a p-value of 0.004 and 0.029 in group A and group B respectively. Thus, both groups are effective in improving HDL levels.

Comparing the two groups was done using the Student's Unpaired t-test, and the results were statistically non-significant before treatment with p-value 0.46 and after completion of treatment statistically significant with p-value 0.028. This shows that both groups are comparable at baseline but group A is more effective than group B in increasing S.HDL levels as shown in table no 06.

In this study, mean value of LDL before treatment was 131.60±47.98 and 146.46±28.60 which was reduced to 117.90±42.05and 125.13±28.81in group A and group B respectively after treatment. LDL showed statistically significant improvement after treatment in both groups with a p-value of 0.0001 so both groups are effective in reducing LDL levels.

Comparing the two groups was done using the Student's Unpaired t-test, and the results were statistically non-significant before and after completion of treatment with p-value 0.15 and 0.44 respectively, that is both trial and study groups are similar at baseline and have equal efficacy in reducing LDL as shown in table no 07.

In this study, mean value of VLDL before treatment was 44.26±13.96and 44.46±15.26 which was reduced to 37.53±12.07and 38.30±12.50in group A and group B respectively after treatment. VLDL showed statistically significant improvement after treatment in both groups with a p-value of 0.0001so both groups are effective in reducing VLDL levels.

Comparing the two groups was done using the Student's Unpaired t-test, and the results were statistically non-significant before and after completion of treatment with p-value 0.95 and 0.81 respectively, that is both trial and study groups are similar at baseline

and having equal efficacy in reducing VLDL as shown in table no 08.

In this study, mean value of TG before treatment was 222.80±68.98and 221.80±76.50 which was reduced to 187.53±60.59 and 189.70±61.95 in group A and group B respectively after treatment. TG showed statistically significant improvement after treatment in both groups with a p-value of 0.0001 thus both groups are effective in reducing TG levels.

Comparing the two groups was done using the Student's Unpaired t-test., and the results were statistically non-significant before and after completion of treatment with p-value 0.95 and 0.89 respectively, that is both groups are comparable and equally effective in reducing TG as shown in table no 09.

Statistically significant improvement was observed in Serum Lipid Profile (TCH, HDL, LDL, VLDL, TG) in both groups, and both groups are comparable at baseline and are equally effective in improving serum lipid levels after treatment except S.HDL. Patients administered *Kulttha Gutika* in group A showed better improvement than patients treated with Atorvastatin in Group B in increasing S.HDL.

Discussion

The Randomized Trial was conducted to assess "Comparative evaluation of Efficacy of *Kulattha Gutika* and Atorvastatin in the Management of Dyslipidemia (*Medoroga*)". *Medoroga* is considered *Santarpanottha Vikara* according to *Ayurveda*.

Lack of physical activity, daytime sleep, and intake of *Kapha* aggravating food leads to the formation of *Madhur Ahar Ras.* This causes an excessive increase in *Sneha* and *Meda* leading to *Mandagni*(27) and *Ama* (Partially digested food) formation. *Strotorodha* caused due to *Ama,* which prevents *Vayu* from moving normally. *Medodhatvagnimandya* inhibits the nutrition of succeeding *Dhatus, this produces* an excessive amount of *Abaddha Meda Dhatu*, which accumulates in the body and causes *Medoroga*(28).

Probable mode of action of Kulattha Gutika

Kulattha Gutika is made up of *Kulattha* as per the standard operating procedure mentioned in *Sharangdhar Samhita*. *Agnimandya*, aggravation of *Kaphavata, Abaddha Meda,* as well as *Strotorodha,* are involved in the pathogenesis of *Medoroga*(29).

Kulattha possesses *Katu, Tikta, Kashaya Rasa, Laghu, Tikshna, Ruksha, Ushna Guna, Katu vipaka,* and *Ushna Virya*. *Tikta*, *Katu Rasa*, and *Ushna Virya* have *Agni* and *Vayu Mahabhoot* predominance which made these drugs *Vatakaphahar, Strotoshodhaka,* and *Vatanuloman. Lekhana, Rukshana* and *Shoshana* properties of *Kulattha* are due to its *Kashaya Rasa and Ushna, Tikshna, Ruksha Guna* that help in reducing excessive *Meda, Kapha,* and *Kleda.* In *Samprapti* of *Medoroga,* there is *Kapha-Vata* dominance, *Agnidushti*, *Stotorodha*, *Vimargagaman,* and *Abaddha Medavruddhi*. Aggravated *Vata* and *Kapha Dosha* are alleviated by the *Vatakaphaghna* property of *Kulattha.* The *Strotoshodhaka* property aids in clearing a path. The *Vatanuloman* property aids in restoring *Koshtagata Vayu's* normal movement. The scraping action caused by the *Lekhana* results in a decrease in the amount of *Abaddha Meda, Kapha,* and *Kleda.* All these properties cause breaking the process of the pathogenesis of *Medoroga.*

Various research studies on phytochemicals stated the presence of active ingredients having antihyperlipidemic and antiobesity actions in *Kulattha*(30). In an animal study conducted by Muthu AK et,al. it was found that a higher dose of the methanolic extract of *Dolichos biflorus* (400 mg/kg body weight) showed comparable results with the standard drug Atorvastatin. Therefore, it can be concluded that the *Dolichos biflorus* possesses hypolipidemic activity in high-fat diet rats(31).

The lipid-lowering effect of *Kulattha* may be due to the presence of phytoconstituents (flavonoids, polyphenols, beta-sitosterol, aminoacids-glycine, isoflavones, isoferririn, cumesterol) (32). This may be brought on by their suppression of hepatic cholesterogensis or by the excretion of faecal sterols.

Kulattha could enhance the activity of the liver`s microsomal cytoplasm p450- dependently hydrolase, which is thought to be involved in the hydroxylation of endogenous steroids like cholesterol (33). This would accelerate the catabolic conversion of cholesterol to bile acid in the liver (34).

In Dyslipidemia, an increase in free fatty acids results from by oxygen-derived free radicals or perhaps by an increase in phospholipase activity, that breaks down membrane phospholipids(35). Due to its ability to eliminate free radicals, chelate metal catalysts, activate antioxidant enzymes, reduce tocopherol radicals, and inhibit oxidase or phospholipase A. *Kulattha* reduced the quantity of free fatty acids(36).

Kulattha decreased the level of triglycerides by reducing activity of lipoprotein lipase in adipose tissue (37). Increased plasma triglyceride uptake by skeletal muscle and adipose tissue may be caused by enhancing the actions of adipose tissue hormone-sensitive lipase and skeletal muscle lipoprotein lipase (38).

Thus, all these activities help in breaking the pathogenesis of Dyslipidemia and showed improvement in serum lipid levels.

Conclusion

From this study, it can be concluded that-

- It is prevalent in *Kapha-Vata Prakruti* with *Manda Agni*.
- Statistically significant improvement was observed in objective parameters like TCH, HDL, LDL, VLDL, and TG in both groups. But in BMI Statistically significant improvement was observed in group A, treated with *Kulattha Gutika*.
- Comparison of both the groups demonstrated no statistically significant difference in objective parameters like TCH, LDL, VLDL, TG, and BMI. Hence both groups are equally effective in Dyslipidemia (*Medoroga*).
- But regarding HDL levels it was observed that statistically significant difference was observed

between both groups. *Kulattha Gutika* was more effective than Atorvastatin.

- So, it is stated that *Kulattha Gutika* is as efficacious as Atorvastatin in managing Dyslipidemia (*Medoroga*) and is an economical alternative without adverse effects.

Further studies can be conducted on a large number of patients for a longer duration of time to confirm efficacy.

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