ORIGINAL ARTICLE

Therapeutic Evaluation of *Lodhradi Kashaya Ghanvati* in Type II Diabetes Mellitus

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Abstract

Aim: To evaluate the therapeutic effects of *Lodhradi Kashaya Ghanvati* (LKGV) in Type II diabetes mellitus (DM) patients. **Materials and Methods:** Forty-five patients of Type II DM were registered from the Outpatient Department of Rasa Shastra, Sir Sunderlal Hospital, Banaras Hindu University, Varanasi. Patients were randomly divided into three groups for the treatment of three different medicine groups (Group A - trial drug only [LKGV 500 mg TID] before meal; Group B - trial drug [LKGV 500 mg TID] before meal + glimepiride [1 mg OD] after meal; Group C - glimepiride treated [1 mg OD]). The treatment was given for 3 months. Fasting blood sugar, postprandial sugar, serum creatinine, blood urea, lipid profile, and serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase levels were estimated biochemically. **Results:** The results of the present study reveal that LKGV can be safe and effective, alternative or adjuvant to the conventional oral hypoglycemic agent. **Conclusion:** The ayurvedic herbal formulation along with the modern drug is the need of hour to take more benefits for the human society suffering from lifestyle disorders.

Key words: *Ayurveda,* Diabetes mellitus, *Ghanvati*, *Kashaya*

INTRODUCTION

Lodhradi Kashaya has been mentioned in the classical text *Basavrajiyam*. It is indicated in the management of *Madhumeha* (diabetes mellitus [DM]).[1] However, the same formulation has been quoted by *Acharya Charaka* for the treatment of *Kaphaja Prameha* [2] The *Lodhradi Kashaya* contains four herbal ingredients *Lodhra (Symplocos racemosa)*, *Haritaki (Terminalia chebula), Musta (Cyperus rotundus)*, and *Katphala (Myrica esculenta)* in equal quantity. In the development of *Prameha*, *Kapha dosha* has dominant role in the initial progress of the disease. If it remains untreated or the causative factors are not excluded, then the progressive pathology leads to the development of *Madhumeha*. By considering these factors and indications by ancient scholars in both stages of the disease, this formulation has been selected by modifying its dosage from *Kashaya* to *Ghanavati* for the present clinical trial. *Madhumeha* can be considered as DM by different perspectives based on clinical symptoms, and attempts have been made by ayurvedic physicians and researchers to treat these two entities using classical formulations mentioned in *Prameha Chikitsa.*[3] In the 21st century, DM has become a considerable major health problem in developed as well as developing countries, which is severely affecting the social and economic development. Currently, one in eleven people globally has diabetes and about 90% of people with diabetes have Type II diabetes. In addition, every 6 s, one person dies from diabetes around the globe.[4] For example, nearly 22% of people with diabetes in the USA use herbal therapy and about 31% use dietary supplements.[5] By considering these factors, the present study has been conducted.

Human safety trial

To conduct clinical trial study, patients were registered from the Rasa Shastra and Kayachikitsa Outpatient Department, Sir Sunderlal Hospital, Institute of Medical Sciences, Banaras Hindu University, Varanasi. Forty-five patients were selected

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on the basic criteria of clinical symptoms of DM explained in texts as well as biochemical parameters required in DM II. After the diagnosis of patients was confirmed, patients were randomly divided into three groups for treatment of three different medicine groups (Institutional Ethical Committee No. Dean/2012-13/181).

Inclusion criteria of patients

- Age >30 and <70 years
- Male and female
- 8-h fasting blood sugar >126 and ≤250 mg/dl and 2-h postprandial blood sugar >200-≤350 mg/dl.

Exclusion criteria

- Type I DM
- Below 30 years and above 70 years patients
- Fasting blood sugar more than 250 mg/dl and postprandial sugar not more than 350 mg/dl
- Patients having longstanding uncontrolled diabetes complication such as nephropathies, retinopathies, and cardiovascular problem
- Pregnant women and lactating mothers.

Diagnosis of the patients

Patients were diagnosed on the basis Type II DM symptoms. Diagnosis of patients was confirmed by estimating fasting blood sugar level and postprandial blood sugar level. Hemoglobin %, serum creatinine, blood urea, lipid profile, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT) were also estimated biochemically.

Grouping of the patients

All the 45 patients were registered for the study after clinical and biochemical examination. Out of them, 38 patients turned up for the full follow-up (FU). All the diagnosed patients were divided into three groups as mentioned in Table 1.

Diet schedule

All selected patients were advised to take less amount of food having carbohydrate and fats.

Criteria to assess the effect of trial drugs

All the selected patients have been advised to come for the FU after every 15 days interval for general examination and estimation of 8-h fasting blood glucose level and 2-h post meal blood glucose level on 1-month interval in three FUs for 90 days. Symptomatic relief along with feeling of well-being to the patients was noted every 15 days in four FUs. Other biochemical parameters of patient were examined before treatment (BT) and after treatment (AT) for the safety of drugs given to the patients and other beneficial effects on the biochemical parameter of diseased patients. Assessments have been done under two headings as subjective assessments and objective assessments.

Subjective assessment

In each FU, the patients were assessed for the subjective improvement of the clinical symptoms, i.e., polyuria, polyphagia, polydipsia, exhaustion/tiredness, and tingling sensation.

Objective assessments

Under the objective parameters, biochemical and other findings have been adopted as follows.

- 8-h fasting and 2-h post meal sugar have been done in three FUs to complete study for 90 days
- Lipid profile was done in patients BT and AT, i.e., 0 day BT and 90 days AT
- Blood urea was done BT and AT (90 days)
- Serum creatinine level was done BT and AT.
- SGOT and SGPT were done BT and AT.

Statistical analysis

The analysis of data was done using statistical software SPSS version 16.0. The collected data were transferred on master chart showing various items/variables in columns and participants in rows.

RESULTS

In the present study, the results of trial drug were assessed under subjective and objective assessments.

Table 1: Grouping of the patients and treatment schedule

Group	Number of patients	Intervention	Vehicle (Anupana)	Dose	Duration and time schedule
A	15	LKGV	Warm water	500 mg	TID before meal
B	15	LKGV+Glimepiride	Warm water	500 mg+1 mg	TID before meal
C	15	Glimipiride	Water	1 mg	OD before meal

LKGV: *Lodhradi Kashaya Ghanvati*

Subjective assessment

The comparison done with respect to the symptom polyuria showed that all groups showed improvement after intervention has given. Group A, Group B, and Group C have the statistically significant results as the *P* value is <0.05. In Group A, 33.3% of patients have no polyuria symptom, 25% have mild symptoms of polyuria, and 25% have moderate symptoms of polyuria. 16% patients of this group presented with severe-grade polyuria symptom. After giving intervention, 66% patients were relieved from polyuria symptom. In Group B, 85.7% of patients had no polyuria symptoms AT, which was 35.7% BT. Similarly, in Group C, there is improvement from 25% to 58.3% in the symptom of polyuria [Table 2].

The comparison done with respect to the symptom polyphagia showed that all groups showed improvement after intervention has given. Group A, Group B, and Group C have statistically significant results as the $P < 0.05$. In Group A, initially 33.3% had no symptom of polyphagia. AT, 66.7% of patients were getting improved from polyphagia symptom. In Group B, there was improvement from 50% to 92.9%, while Group C showed improvement from 33.3% to 83.7% in polyphagia symptom [Table 3].

The comparison done with respect to the symptom polydipsia showed that all groups showed improvement after intervention has given. Group A and Group B have the statistically significant results as the $P < 0.05$. Group C was statistically not significant. In Group A, 50% of patients have no symptom of polydipsia. AT, 66.7% of patients were got improved. In Group B, there was improvement from 57.1% to 85% in symptom polydipsia [Table 4].

The comparison done with respect to the symptom exhaustion/tiredness showed that all groups showed improvement after intervention has given. Group A and Group C have the statistically significant results as the $P < 0.05$, while Group B showed statistically highly significant result as $P < 0.001$. In Group A, initially, 33.3% had no symptom of tiredness. AT, 75% of patients were getting improved from tiredness symptom. In Group B, there was improvement from 14.3% to 92.9%, while Group C showed improvement from 16.7% to 66.7% in tiredness symptom [Table 5].

The comparison done with respect to the symptom tingling sensation showed that all groups showed improvement after intervention has given. Group A, Group B, and Group C have the statistically significant results as the $P < 0.05$. In Group A, initially, 25% had no symptom of tiredness. AT, 50% of patients were getting improved from tiredness symptom. In Group B, there was improvement from 50% to 58.3%, while Group C showed improvement from 33.3% to 58.3% in tingling sensation symptom [Table 6].

Objective assessment

Effect of interventions on fasting blood glucose and postprandial blood glucose in different FU among study groups [Figures 1 and 2]

The comparison done with respect to the mean fasting and mean postprandial blood glucose level showed that all groups showed improvement after intervention has given. The mean fasting blood glucose level decreased in successive FUs as compared to initial. Group B and Group C were statistically

Table 2: Effect on polyuria symptom in different FU among study groups

Groups	Grade	FU of patients (polyuria), *N* (%)				
		BT	FU_1	FU_2	FU_3	Wilcoxon-signed rank test
A	0	4 (33.3)	4 (33.3)	6 (50.0)	8 (66.7)	*Z*=2.640 *P*=0.008
	1	3 (25.0)	3 (25.0)	3 (25.0)	3 (25.0)	
	2	3 (25.0)	3 (25.0)	3 (25.0)	1 (8.3)	
	3	2 (16.0)	2 (16.7)	0 (0)	0 (0)	
B	0	5 (35.7)	6 (42.9)	8 (57.1)	12 (85.7)	*Z*=2.739 *P*=0.006
	1	4 (28.6)	5 (35.7)	5 (35.7)	2 (14.3)	
	2	4 (28.6)	2 (14.3)	1 (7.1)	0 (0)	
	3	1 (7.1)	1 (7.1)	0 (0)	0 (0)	
C	0	3 (25.0)	3 (25.0)	7 (58.3)	7 (58.3)	*Z*=2.733 *P*=0.05
	1	4 (33.3)	6 (50.0)	5 (41.7)	5 (41.7)	
	2	5 (41.7)	3 (25.0)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	

N: Number of patients, Values are statistically significant at **P*<0.05, ***P*<0.001. BT: Before treatment, FU: Follow-up. Group A, Group B, and Group C have the statistically significant results as the P value is <0.05.

Table 3: Effect on polyphagia symptom in different FUs among study groups

Groups	Grade	FU of patients (polyphagia), *N* (%)				
		BT	FU_1	FU_2	FU_3	Wilcoxon-signed rank test
A	0	4 (33.3)	4 (33.3)	7 (58.3)	8 (66.7)	*Z*=2.640 *P*=0.008
	1	4 (33.3)	4 (33.3)	3 (25)	4 (33.3)	
	2	2 (16.7)	4 (33.3)	2 (16.7)	0 (0)	
	3	2 (16.7)	0 (0)	0 (0)	0 (0)	
B	0	7 (50)	8 (57.1)	11 (84.6)	13 (92.9)	*Z*=2.530 *P*=0.011
	1	5 (35.7)	5 (35.7)	2 (15.4)	1 (7.1)	
	2	2 (14.3)	1 (7.1)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	
C	0	4 (33.3)	4 (33.3)	9 (75)	10 (83.7)	*Z*=2.646 *P*=0.008
	1	7 (58.3)	8 (66.3)	3 (25)	2 (16.7)	
	2	1 (8.3)	0 (0)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	

N: Number of patients. Values are statistically significant at **P*<0.05, ***P*<0.001. BT: Before treatment, FU: Follow up. Group A, Group B, and Group C have statistically significant results as the *P*<0.05.

Table 4: Effect on polydipsia symptom in different FUs among study groups

Groups	Grade	FU of patients (polydypsia), *N* (%)				
		BT	FU_1	FU_2	FU_3	Wilcoxon-signed rank test
Group A	0	6 (50)	6 (50)	6 (50)	8 (66.7)	*Z*=2.271 *P*=0.023
	1	1 (8.3)	1 (8.3)	4 (33.3)	3 (25)	
	2	3 (25)	3 (25)	2 (16.7)	1 (8.3)	
	3	2 (16.7)	2 (16.7)	0 (0)	0 (0)	
Group B	0	8 (57.1)	9 (64.3)	11 (78.6)	12 (85)	*Z*=2.236 *P*=0.025
	1	5 (35.7)	4 (28.6)	3 (21.4)	2 (14.3)	
	2	1 (7.1)	1 (7.1)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	
Group C	0	4 (33.3)	4 (33.3)	6 (50)	8 (66.7)	*Z*=1.66 *P*=0.096
	1	7 (58.3)	8 (66.7)	6 (50)	4 (33.3)	
	2	1 (8.3)	0 (0)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	

N: Number of patients, Values are statistically significant at **P*<0.05, ***P*<0.001. BT: Before treatment, FU: Follow up. Group A and Group B have the statistically significant results as the P < 0.05. Group C was statistically not significant.

highly significant. The mean postprandial blood glucose level decreased in all groups at 6th FU as compared to initial. Group A, Group B, and Group C were statistically highly significant [Figure 3 and Table 7].

The mean blood urea decreased AT in Group A and B was 4.04 and 3.43, respectively. Group A was statistically significant. In Group C, increased mean blood urea was observed [Figure 4 and Table 8].

The comparison done with respect to mean serum creatinine showed that all groups showed improvement after intervention has given. The decrease in mean serum creatinine AT was statistically significant in Group A as $P < 0.05$ [Figure 5 and Table 9].

The comparison done with respect to mean SGOT and mean SGPT showed that all groups showed improvement after intervention has given, expect Group C which showed

Table 5: Effect of interventions on exhaustion/tiredness in different FU among study groups

Groups	Grade	FU of patients (exhaustion/tiredness), *N* (%)				
		BT	FU_1	FU_2	FU_3	Wilcoxon-signed rank test
A	0	4 (33.3)	4 (33.3)	8 (66.7)	9 (75)	*Z*=2.271 *P*=0.023
	1	5 (41.7)	6 (50)	4 (33.3)	3 (25)	
	2	3 (25)	2 (16.7)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (71.4)	0 (0)	
B	0	2 (14.3)	4 (28.6)	10 (21.4)	13 (92.9)	*Z*=3.357 *P*=0.001
	1	10 (71.4)	9 (64.3)	3 (7.1)	1 (7.1)	
	2	2 (14.3)	1 (7.1)	1 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	
C	0	2 (16.7)	3 (3)	6 (50)	8 (66.7)	*Z*=3.00 *P*=0.003
	1	7 (58.3)	8 (8)	6 (50)	4 (33.3)	
	2	3 (25)	1 (1)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	

N: Number of patients. Values are statistically significant at **P*<0.05, ***P*<0.001. BT: Before treatment, FU: Follow up. Group A and Group C have the statistically significant results as the P < 0.05, while Group B showed statistically highly significant result as *P*<0.001.

Table 6: Effect on tingling in different follow-ups among study groups

Groups	Grade	FU of patients (tingling), *N* (%)				
		BT	FU_1	FU_2	FU_3	Wilcoxon-signed rank test
A	0	3 (25)	3 (25)	5 (41.7)	6 (50)	*Z*=2.828 *P*=0.005
	1	4 (33.3)	4 (33.3)	5 (41.7)	5 (41.7)	
	2	4 (33.3)	4 (33.3)	2 (16.7)	1 (8.3)	
	3	1 (8.3)	0 (8.3)	0 (0)	0 (0)	
B	0	7 (50.0)	7 (53.8)	10 (71.4)	10 (58.3)	*Z*=2.00 *P*=0.044
	1	6 (42.9)	5 (38.5)	4 (28.6)	4 (41.7)	
	2	1 (7.1)	1 (7.7)	0 (0)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	
C	0	4 (33.3)	5 (41.7)	7 (58.3)	7 (58.3)	*Z*=2.00 *P*=0.044
	1	7 (58.3)	6 (50)	4 (33.3)	5 (41.7)	
	2	1 (8.3)	1 (8.3)	1 (8.3)	0 (0)	
	3	0 (0)	0 (0)	0 (0)	0 (0)	

N: Number of patients. Values are statistically significant at **P*<0.05, ***P*<0.001. BT: Before treatment, FU: Follow-up. Group A, Group B, and Group C have the statistically significant results as the P < 0.05.

increase in mean SGOT AT. Group A found statistically significant in decreasing mean SGPT. In case of mean SGPT, all groups showed not statistically significant result [Figure 6 and Table 10].

The mean total cholesterol (TC) decreased cholesterol AT in Groups A and B was 18.41 and 14.21, respectively, and both were statistically highly significant. However, in Group C, minor increased mean value was observed. The mean triglyceride (TG) decreased AT in Groups A and B was 18.33 and 9.86, respectively. Group B was found statistically highly significant. Increased mean TG was observed in Group C. The mean high-density lipoprotein (HDL) increased AT in Group A and B was 3.08 and 2.0, respectively. Group A was statistically highly significant while Group B also found statistically significant. In Group C, minor decrease in mean HDL value was observed. The mean low-density lipoprotein (LDL) decreased AT in Groups A and B was 12.83 and 10.42,

Table 7: Effect of interventions on fasting blood glucose and postprandial blood glucose level BT and AT

Groups	Mean±SD			
	Fasting blood sugar		Postprandial blood sugar	
	BT	AT	BT	AT
A	172.33±18.23	145.42±58.05	271.83±44.43	168.58±29.90**
B	183.93±13.60	111.36±10.14**	259.79±55.25	147.00±38.53**
C	174.08±18.23	105.08±9.130**	282.08±41.32	139.89±10.53**

Values are statistically significant at *P<0.05, **P<0.001. BT: Before treatment, AT: After treatment, SD: Standard deviation. Group B and Group C were statistically highly significant in reducing the Fasting blood sugar while Group A, Group B and Group C were showing statistically highly significant improvement in case of Post prandial blood sugar level.

Table 8: Effect of interventions on blood urea BT and AT

Groups	Blood urea (mean±SD)	
	BT	AT
A	19.29±9.81	15.25±7.55*
B	23.07±11.30	19.64±7.41
C	21.66±9.03	23.58±9.94

Values are statistically significant at *P<0.05, **P<0.001.
BT: Before treatment, AT: After treatment, SD: Standard deviation.
The statistically significant result was observed in Group A.

Table 9: Effect of interventions on serum creatinine BT and AT

Groups	Serum creatinine (mean±SD)	
	BT	AT
A	0.808±0.215	0.625±0.176*
B	0.664±0.324	0.628±0.226
C	0.808±0.267	0.800±0.225

Values are statistically significant at *P<0.05, **P<0.001.
BT: Before treatment, AT: After treatment, SD: Standard deviation.
In Group A , statistically significant result was observed.

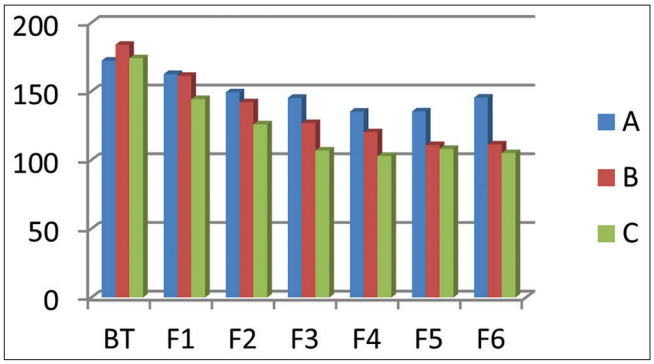

Figure 1: Fasting blood glucose level

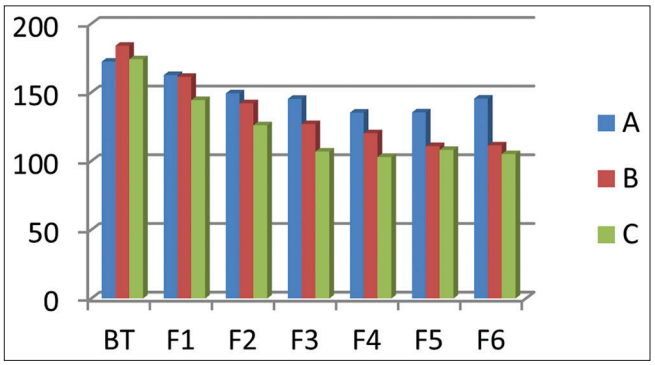

Figure 2: Postprandial blood glucose

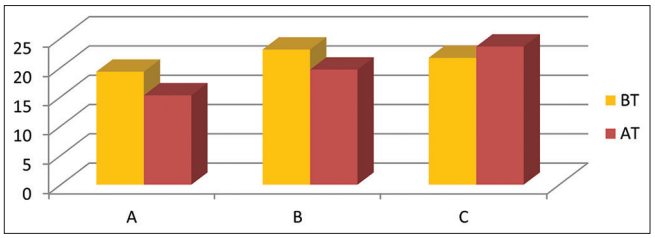

Figure 3: Blood urea level before and after trial in various study groups

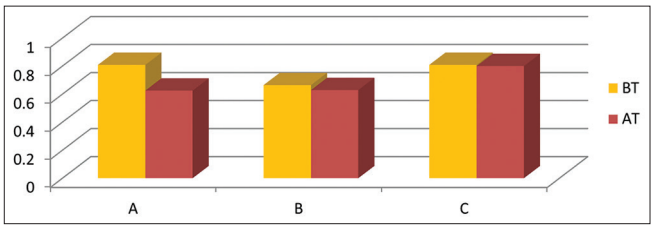

Figure 4: Serum creatinine level before and after trial in various study groups

respectively, and Groups A and B were found statistically highly significant. However, in Group C, increased mean LDL was observed. The mean very LDL (VLDL) decreased AT in Groups A and B was 2.83 and 2.64, respectively, and Group A was statistically highly significant. Furthermore, Group B was statistically significant. However, in Group C, increased mean VLDL was observed [Table 11].

DISCUSSION

Herbal medicines have great demand in the developed and developing countries for primary healthcare because of their wide biological and medicinal activities, higher

Table 10: Effect of interventions on SGOT and SGPT BT and AT

Groups	Mean±SD			
	SGOT		SGPT	
	BT	AT	BT	AT
A	37.65±24.23	26.75±9.47*	38.75±17.57	32.25±8.368
B	36.57±29.09	31.36±14.89	42.64±27.20	28.79±10.36
C	18.75±6.01	21.92±4.85	32.17±7.17	29.42±3.343

Values are statistically significant at *P<0.05, **P<0.001. BT: Before treatment, AT: After treatment, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, SD: Standard deviation. In case of SGOT Group A was observed statistically significant. While all the three groups have shown statistically non significant results in case of SGPT.

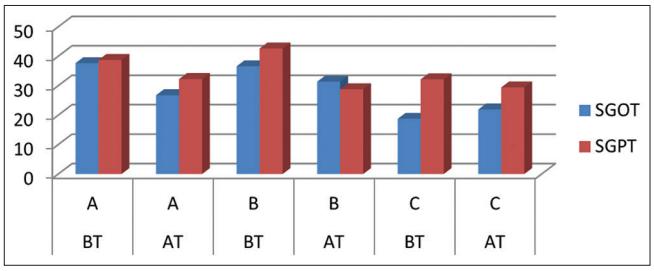

Figure 5: Serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase level before and after trial in various study groups

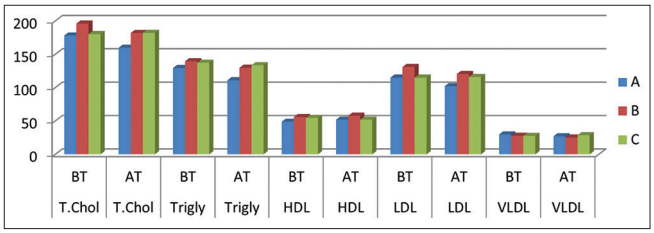

Figure 6: Effect of trial treatment on lipid profile before trial and after follow-ups in various study groups

safety margins, and lesser cost. Even in the 21st century, no specific treatments are available for the management and/or prevention of DM. Various scientific works proving the antidiabetic activities of all the four ingredients of *Lodhradi Kashaya Ghanvati* (LKGV) have been published.

It has been observed that methanolic extract of *M. esculenta* leaves was found more effective in the treatment of diabetes and showed significant antidyslipidemic effect in streptozotocin (STZ)-induced diabetes in rats.[6] The increased levels of plasma glucose in STZ-induced diabetic rats were lowered by the administration of *S. racemosa* Roxb. The reduced glucose levels suggested that *S. racemosa* Roxb. might exert insulin-like effect on peripheral tissues by either promoting glucose uptake metabolism by inhibiting hepatic gluconeogenesis[7,8] or by absorption of glucose into the muscle and adipose tissues,[9] through the stimulation of a regeneration process and revitalization of the remaining beta-cells.[10-12] The methanolic extract of *S. racemosa* (MESR) exhibited hypocholesterolemic and hypotriglyceridemic effects, while increased the levels of HDL in STZ-induced diabetic rats. However, MESR was found to be more effective in reducing the levels of TG and LDL as compared to its effect on TC.[13] MESR at tested doses produced a significant reduction ($P < 0.01$) in the carbon tetrachloride (CCl_4)-induced elevated levels of SGOT, SGPT, alkaline phosphatase, gamma glutamyltransferase, and total bilirubin as well as increased the total protein when compared to the animals treated only with CCl_4 after 36 h of CCl_4 treatment. Overall, MESR at tested doses significantly reduced the levels of hepatic enzymes and total bilirubin.[14] The present herbal classical formulation LKGV has been trialed in the experimental study where the formulation proved its antihyperglycemic effect in STZ-induced diabetic rats.[15]

In the present study, from the result, it is quite evident that LKGV is effective in patients with Type 2 DM. In case of classical symptoms of DM, i.e., subjective assessment [Tables 2-6], the formulation showed statistically significant results alone (Group A) as well as with modern drug (Group B). This improvement is may be due to glycemic control in diabetic patients. Table 6 shows the effect of test drug on fasting as well as postprandial blood sugar level where Group B and Group C show statistically highly significant result. The effect of formulation on blood urea and serum creatinine shows that LKGV (Group A) has statistically significant result than along with glimepiride (Group B) and comparative drug glimepiride only (Group C). Liver enzymes also get disturbed in DM. In the present study, Table 10 shows that Group A found statistically significant in decreasing mean SGPT while glimepiride-treated group (Group C) showed slight increase in mean SGOT. It may be due to liver protecting activity of LKGV. Lipid metabolism is also disturbed in DM. The effect of test formulation LKGV is shown in Table 11. Group A and Group B showed statistically significant results in case of lipid profile. This is may be due to the ingredients of LKGV increasing the excretion of fecal bile acids and neutral steroids, which lower the cholesterol levels in the blood and other tissues or by some unknown mechanism.

CONCLUSION

On the basis of the present study, it could be concluded that the LKGV exhibited antihyperglycemic and antihyperlipidemic

Table 11: Effect of interventions on lipid profile BT and AT

Group	Lipid profile (mean±SD)											
	TC		TG		HDL		LDL		VLDL			
	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT		
A	178.17±35.32	159.75±28.51**	129.33±16.27	111.00±33.54	48.83±7.62	51.92±7.16**	115.00±14.68	102.17±10.67**	29.92±6.52	27.08±4.10**		
B	196.07±20.14	181.86±17.84**	139.57±9.96	129.71±10.46**	55.83±5.72	57.86±3.84*	130.86±16.46	120.43±13.88**	27.79±6.60	25.14±4.60*		
C	180.25±22.46	182.00±21.42	137.25±13.12	133.50±10.69	54.50±6.08	52.00±5.72**	114.92±18.40	116.00±15.937	27.67±5.75	28.75±3.54		

Values are statistically significant at *$P<0.05$, **$P<0.001$. BT: Before treatment, AT: After treatment, TC: Total cholesterol, TG: Triglyceride, LDL: Low-density lipoprotein, HDL: High-density lipoprotein

activity for which significant improvement in symptoms and sign were observed and significant euglycemia was attained. The LKGV along with modern drug glimepiride has promising results. The ayurvedic herbal formulation along with the modern drug is the need of hour to take more benefit for the human society suffering from lifestyle disorders.

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