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Original Research Article

Effect of herbal combination of triphala and *Garcinia cambogia* extracts on liver function test and kidney function test in high fat diet induced obesity in rats

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ABSTRACT

Background: Obesity, a global epidemic, is a major risk factor for diabetes mellitus and cardio vascular diseases. Despite advances, the pharmacotherapy for obesity remains limited. Almost all medications for long term management of obesity have health issues. Due to the adverse drug reactions (ADRs) associated with many antiobesity medicines, the clinical trials are focussing on screening herbal medicines for use in the treatment of obesity, which have minimal ADRs.
Methods: Rats were divided into eight groups of six each. The rats were first made obese by feeding high fat diet (HFD) for three weeks. Then treatment with the herbal extracts was given simultaneously with the HFD to the experimental groups. Rats were fed HFD for six weeks along with herbal extracts and the effect on their liver function test and kidney function test were evaluated.
Results: The rats fed HFD and supplemented with herbal preparations of Triphala and *G. cambogia* for six weeks, showed significant improvement in liver function test and kidney function test related parameters as compared to the control group rats fed with HFD alone.
Conclusions: Triphala and *G. cambogia* can counter the effects of HFD intake and have the potential for use as anti-obesity agents with desirable liver function test and kidney function test related parameters modulating properties.

Keywords: Obesity, Triphala, *Garcinia cambogia*

INTRODUCTION

Obesity is a medical condition in which excess body fat is accumulated to the extent that it may harm health.[1] Obesity is a risk factor for non-insulin-dependent diabetes mellitus (NIDDM), cardio vascular disease (CVD), osteoarthritis, cancer, reproductive and metabolic disorders.[2] It is also associated with disturbance in the hormonal milieu that can affect the reproductive system in women who present reproductive disorders when obese.[3,4] In humans, this relationship is poorly characterized, due to limited studies.[5] Some energy balance genes are essential for normal regulation and mutation in a single gene can lead to obesity in lab animals.[6] However, this does not explain obesity in the majority of the human population where no such genetic changes are seen. Concomitant diets are a major factor in the current obesogenic environment, and human obesity is diet-induced.[6] Although genetic obesity models are useful in finding the role of endogenous neuropeptides in

body weight control, the best parallels to human obesity are provided by the physiological model of diet-induced obesity.[6,7]

The causative factors of obesity are multiple and complex. The influence of the environment also exists, since the human genotype has not changed substantially in past 30 years. Thus, small changes in daily lifestyle, such as the use of machines for washing clothes and dishes and automobiles for transportation have a significant impact on the total daily energy spent. In addition to the total energy use due to low physical activity, environmental factors contribute to greater energy intake, through excess fat in the diet, consumption of high-calorie food, large-sized portions, frequency of food intake, lower cost and greater availability of food.[8] The increased storage that creates obesity eventually leads to the release of excessive fatty acids from enhanced lipolysis, which is stimulated by the enhanced sympathetic state in obesity.[9] The release of these excessive free fatty acids, lipids and their metabolites, creates oxidative stress to the endoplasmic reticulum and mitochondria. This affects adipose as well as non-adipose tissue.[10]

The high prevalence of overweight and obesity, conjoint with their concomitant health risks, makes it particularly relevant worldwide public health challenge. Global projections estimate 1.12 billion individuals to be obese by the year 2030 and this rapid growth of obesity will occur in adults and children alike.[11,12] Increase in weight and obesity are attributable to a global shift in diet towards increased intake of energy-dense foods and a trend towards less physical activity. Obesity is a major contributor to morbidity and mortality, surpassing drinking and smoking in negative effects on health. This will have negative effects on life expectancies of generations born after the rise of the obesity epidemic.[13] The theories overlap. Firstly, obesity has been viewed as a disease of energy balance due to an excess intake and decreased expenditure. Attention has been focused on the mechanisms controlling feeding behaviour, food intake and adipose mass.[15]

Secondly, obesity is seen as a disorder of the adipocytes as it has a mechanism of fat storage and mobilization. With growing understanding, the traditional view of adipose tissue has switched from being a passive energy "reservoir" with insulator attributes to a complex, highly active and essential metabolic and endocrine organ, churning out an assortment of hormones and other signals regulating the body physiology (e.g., food intake, body weight and brain activity).[16] It has been recognized as having an independent endocrine role that can result in an inflammatory response with increased risk of type 2 diabetes mellitus and cardio vascular disease (CVD), leading to increased morbidity and early mortality.[17] Adipose tissue affects energy homeostasis and CV health by releasing adipokines that regulate energy expenditure, food intake, insulin sensitivity and inflammation.[17] Thirdly, obesity is a neurobehavioral disorder with the control of appetite and food intake involved in obesity pathogenesis.[14,17,18] Obesity is associated with insulin resistance, i.e., there is suppressed or delayed response to insulin. Hormones, cytokines and metabolic fuels from the adipocyte diminish insulin action. Large adipocytes in obese are resistant to insulin suppression of lipolysis, especially in visceral fat. These all result in increased levels of fatty acids and glycerol, which aggravate insulin resistance in skeletal muscles and liver.[19] Obesity itself may also induce systemic oxidative stress and that increased oxidative stress in accumulated fat at least in part, the underlying cause of dysregulation of adipo-cytokines and development of metabolic syndrome.

Table 1: Mechanism of bioactive compounds of triphala.

Herbal plant	Bioactive compounds	MOA
Terminalia bellerica	Tannins, quinines, phenols, coumarines, flavanoids and phytosterols	Reduce lipids in serum.[22]
Emblica officinalis	Flavonoids	Improves lipid profiles by inhibiting synthesis of cholesterol via decreasing hepatic 3-hydroxy-3-methyl glutaryl-coenzyme A (HMG-CoA) reductase and also enhances degradation of cholesterol. [23]
	Gallic acid, gallotanin, ellagic acid, corilagin	Antiobesity, anti-inflammatory, antidiabetic effects through their antioxidant and free radical scavenging properties.[23-26]
Terminalia chebula	Gallic acid, methyl gallate, ethyl gallate, chebulagic acid, tetra-*O*-galloyl-*β*-D-glucose, ellagic acid, chebulinic acid and penta-*O* galloyl-*β*-D-glucose, phenolic compounds, punicalagin, Terflavin-A, Terchebulin Girin.	Antiobesity, hypolipidemic effect.[27,28]

Table 2: Mechanism of bioactive compounds of *Garcinia cambogia.*

Herbal plant	Bioactive compounds	MOA
Garcinia cambogia	HCA	Inhibits the enzyme ATP-citrate lyase, which is involved in endogenous lipid biosynthesis.[29-31]
	HCA	In addition, there is an increased production of hepatic glycogen in the presence of HCA, which may activate glucoreceptors leading to a sensation of fullness and reduced appetite.[29-31]
	HCA	The increased bioavailability of serotonin is thought to be related to appetite suppressing effects of supplemental HCA.[32]
	HCA	Another possible MOA may be HCA's ability to down-regulate Leptin, an amino acid hormone that induces obesity and body weight.[33-37]

Preventive or therapeutic strategies to control obesity should target the abnormalities associated with obesity. Pharmacological intervention includes sibutramine, orlistat, phentermine, diethylpropion, and fluoxetine or bupropion. Phentermine and diethylpropion have potential for abuse and are approved for short-term use. Approved medications for long term use in the treatment of obesity are sibutramine and orlistat, however, these should be used with caution in patients with history of CVD due to harmful ADRs associated.[20] Due to the ADRs associated with many of the anti-obesity medicines, recent drug trials have focused on screening the herbal medicines to treat obesity with minimal ADRs. Anti-obesity foods may prevent the condition, possibly leading to the prevention of life style related diseases, if they are effective in reducing the visceral fat mass. This study was undertaken with an aim to evaluate the effect of herbal extracts of triphala, *G. cambogia*, and combination of triphala and *G. cambogia* extracts in an experimental model of high fat diet (HFD) induced obesity in albino Rats, in reducing the visceral fat deposition. [21]

Objectives

The objectives of the present study were to study the effect of aqueous extract of triphala in HFD induced obesity model in rats, to study the effect of aqueous extract of *G. cambogia* in HFD induced obesity model in rats and to study the effect of combination of aqueous extracts of triphala and *G. cambogia* in HFD induced obesity model in rats.

METHODS

Study design

The prospective study was conducted on 48 male albino Wistar rats which weighed between 150-200 grams.

Place of work done

The study was conducted at Centre for Scientific Research and Development (CSRD), People's University, Bhopal.

Herbal extracts for the study

Standardized aqueous extracts of triphala was having tannins 45% and gallic acid by high performance liquid chromatography (HPLC) 7% and *G. cambogia* used contained 60% hydroxy citric acid by HPLC USP method. These extracts procured from Gujarat based certified company, M/s Pharmanza Herbal Pvt. Ltd. The doses were prepared freshly before experimentation.

Materials used

Triphala extract (Amla, Harad and Bahed), *Garcinia cambogia* extract and atorvastatin.

Animal experiments

All the animal experiments were performed in accordance with the protocol approved by the institutional animal ethics committee (IAEC) of People's College of Medical Sciences and Research Centre (PCMSRC), People's University, Bhopal, Madhya Pradesh, India.

Procurement of rats and HFD diet

The animals were purchased from National Institute of Nutrition, Hyderabad, Telangana India.

Approval from IAEC

The approval of IAEC, PCMSRC was obtained dated 30 November 2016.

Housing and feeding conditions

The rats were maintained in appropriate conditions as per the CPCSEA guidelines. The rats were maintained on standard pellet diet normal diet (ND) and HFD *ad libitum* and free access to water.

Experimental diets

Standard rat chow ND and HFD were purchased from National Institute of Nutrition Hyderabad, Telangana.

Description about grouping, dosing and feeding

Animals were divided into 8 groups with each group consisting of six rats and the experimental period was 6 weeks.

- Group 1 was given normal diet and served as control.
- Group 2 was given HFD 45% kcal fat throughout the study period of 42 days.
- Group 3 was given HFD for 21 days then from 22nd day of the study till the end of the study atorvastatin and HFD. This group acted as positive control.
- Group 4 was given HFD for 21 days then from 22nd day of the study till the end of the study triphala extract and HFD.
- Group 5 was given HFD for 21 days then from 22nd day of the study till the end of the study *G. cambogia* extract and HFD.
- Group 6, 7, 8 were given HFD for 21 days then from 22nd day of the study till the end of the study-combination of aqueous extract of *G. cambogia* and triphala at 500, 1000 and 2000 mg/kg respectively and HFD.

Statistical analysis

The data was expressed as mean±SD and analysed by using one-way ANOVA followed by post-hoc test. The values of $p<0.001$ and $p<0.05$ were considered as statistically significant. Statistical software SPSS 25.0 version and statistical functions in MS-Excel 2010 were used.

RESULTS

Liver function tests (LFT)

ALT is a very good indicator enzyme to assess liver toxicity. We found the levels of ALT enzyme to be elevated after induction of obesity by HFD in 21 days, and its level significantly increased from a control value of 13.65±5.06 to 17.55±5.35 U/l. After 21 days of a combination of triphala and *G. cambogia* extract oral administration in male rats the value was 6.08±2.11U/l. With co-treatment of individual extracts of triphala and *G. cambogia*, the ALT levels were found to be 9.95±5.60 U/l, 6.8±4.40 *U/l which is also lower as like that of the combination extract-treated value 6.08±2.77 U/l ($p<0.05$).

AST is a good indicator enzyme to assess liver toxicity. In our study, the levels of AST enzyme were found to be elevated after induction of obesity by HFD in 21 days, where its level increased from a control value of 21.93±6.98 to 26.23±6.65 U/l. After 21 days of a combination of triphala and *G. cambogia* extract oral administration in male rats the value was 8.11±4.27 U/l. With co-treatment of individual extracts such as triphala extract and G. cambogia extract the ALT levels were found to be 20.9±9.13 U/, 11.73±5.16 U/l which is decreased like with the combination extract-treated value 8.11±4.27 U/l but these values did not show any statistically significant difference ($p<0.001$, $p<0.05$). ALP is an important diagnostic tool in hepatitis, biliary obstruction, hyperparathyroidism, etc. The value of alkaline phosphatise in this study was found to be elevated in 21days of HFD group in which the value increased from the control value of 18.50±2.58 to 32.89±5.88 U/l. On the other hand, co-treated rats with individual triphala and *G. cambogia* extracts showed a decreased level of ALP where the values were found to be 36.50±2.58 U/l ($p<0.05$), 29.77±14.78 U/l ($p<0.05$) respectively.

In the combination of triphala and *G. cambogia* treated group 107.31±21.93 U/l ($p<0.001$), all the values of ALP showed statistically significantly difference when the control group was compared to HFD, triphala, *G. cambogia* and the combination of triphala and *G. cambogia*. We investigated the effects of triphala extract, *G. cambogia* extract and combination of both extracts on protein concentration after the HFD induced obesity in male Wistar rats during 42 days of the experimental period. The data indicated that after induction of obesity by HFD in male Wistar rats, the level of total proteins decreased after 21 days, where the value was found to be 7.62±1.06 g/dl ($p<0.05$) which is lower than the control value of 8.58±0.9 g/dl ($p<0.05$). With co-treatment of individual triphala extract and *G. cambogia* extract the total protein was found to be 8.45±0.91 g/dl and 8.17±0.66 g/dl respectively which is almost equal to control group values of 8.58±0.9 g/dl. With co-treatment of the combination of triphala and *G. cambogia* values two deferent doses were 9.08±0.57 ($p<0.05$), 9.66±0.60 g/dl ($p<0.05$) respectively.

Table 3: Effect of drugs on ALT or SGPT.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 day	22.78± 4.50	13.65 ±5.06	6.56± 2.72	12.5± 3.58	8.9± 4.38	18.15± 2.87	6.03± 3.36	8.78± 4.48
21st day	20.23± 6.53	17.55 ±5.35	10.76± 3.73	15.11± 3.20	10.23± 5.04	20.86± 3.38	8.15± 2.44	12.98± 2.66
42nd day	16.21± 6.76	12.23 ±7.02	8.86± 4.48	9.95± 5.60	6.8± 4.40	14.65± 6.13	5.13± 1.72*	6.08± 2.77

Values are mean±SEM; n=6 in each group. All means are statistically significantly different (**: $p<0.001$, *: $p<0.05$, $^{\#}$: $p>0.05$ statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

Table 4: Effect of drugs on AST or SGOT.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 day	32.03± 4.43	21.93± 6.98	20.41± 6.84	25.45± 6.09	9.45± 3.72	11.81± 2.68	10.16± 5.95	13.96± 4.08
21st day	33.56± 4.37	26.23± 6.65	23.63± 7.50	28.96± 5.57	14.55± 5.76	16.55± 4.77	13.76± 5.67	11.28± 5.67
42nd day	23.11± 10.25	17.15± 6.77	16.46± 9.96	20.9± 9.13	11.73± 5.16	12.03± 7.22	11.61± 5.28	8.11± 27

Values are mean ±SEM; n=6 in each group. All means do not show any statistically significant difference (**: p<0.001, *: p<0.05, #: p>0.05 statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

Table 5: Effect of drugs on ALP.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 day	18.50± 2.58	23.05± 3.25	30.91± 6.10	40.63± 5.83	60.97± 8.20	116.65± 5.13	66.41± 6.52	116.1± 6.26
21st day	50.39± 34.85	32.89± 5.88*	33.15± 9.46	31.80± 4.88	31.15± 15.55	166.36± 13.42	71.81± 8.58	121.32± 7.10
42nd day	38.92± 26.57	29.77± 6.86	35.41± 3.54	36.50± 2.58*	29.77± 14.78*	148.75± 18.14*	69.65± 6.30	107.31± 21.93**

Values are mean ±SEM; n=6 in each group. All means are statistically significantly different (**: p<0.001, *: p<0.05, #: p>0.05 statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

Table 6: Effect of drugs on total protein.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 day	8.58± 0.9	7.06± 1.42	7.14± 1.38	7.09± 1.64	6.88± 0.86	6.66± 1.25	7.89± 1.49	7.06± 1.31
21st day	7.87± 0.86	7.62± 1.06*	7.46± 1.02	8.14± 1.03	7.71± 0.77	7.56± 098	8.58± 0.90	8.47± 0.99
42nd day	7.65± 0.80	7.88± 1.05	7.95± 0.78	8.45± 0.91	8.17± 0.66	8.26± 1.10	9.08± 0.57*	9.66± 0.60*

Values are mean±SEM; n=6 in each group. All means are statistically significantly different (**: p<0.001, *: p<0.05, #: p>0.05 statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

Table 7: Effect of drugs on albumin.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 Day	4.62± 0.66	4.59± 0.91	5.23± 1.11	3.85± 0.89	3.87± 0.48	4.05± 0.84	4.52± 1.18	4.06± 1.28
21st Day	4.52± 0.60	4.90± 0.54*	5.78± 0.98	4.11± 0.96	4.74± 0.56	4.54± 1.38	5.15± 0.96	5.54± 0.88
42nd Day	4.26± 0.66	3.75± 0.78	4.9± 0.96	3.40± 0.70	3.95± 0.69	3.58± 1.46	3.89± 1.07	2.94± 0.88**

Values are mean±SEM; n=6 in each group. All means are statistically significantly different (**: p<0.001, *: p<0.05, #: p>0.05 statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

We found that with a combination of triphala and *G. cambogia* treatment for 21 days, the total protein levels slightly increased as compared to the control group. We investigated the effects of triphala extract, *G. cambogia* extract and combination of both extracts on albumin concentration after the HFD induced obesity in male Wistar rats during 42 days of the experimental period. The data indicated that after induction of obesity by HFD in male Wistar rats, the level of albumin increased after 21 days, where the value was found to be 4.90±0.54 g/dl which is higher than the control value of 4.62±0.66 g/dl ($p<0.05$). With co-treatment of individual triphala extract and *G. cambogia* extract, the albumin value was found to be 3.40±0.70 g/dl and 3.95±0.69 g/dl respectively, which is lower as compared to combination-treated value 1.94±0.88 g/dl ($p<0.001$).

Our data shows that the combination of triphala and *G. cambogia* was effective than the individual herbal treatment.

Higher levels of bilirubin indicate different types of liver problems. Occasionally, higher bilirubin levels may indicate an increased rate of destruction of red blood cells (hemolysis). In our study we investigated the effects of triphala extract, *G. cambogia* extract and combination of both extracts on bilirubin concentration after the HFD induced obesity in male Wistar rats during 42 days of the experimental period.

Table 8: Effect of drugs on bilirubin.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 day	210.5± 48.06	156.48 ±46.36	181.33± 31.97	158± 23.39	242.16± 30.28	253± 16.39	273.41± 29.88	276± 18.52
21st day	200.62± 55.91	167.75 ±42.93	193.34± 32.82	167.61± 27.19	252.22± 34.57	277.27± 22.05	317.12± 42.72	311.02± 44.28
42nd day	206.72± 33.10	165.66 ±41.75	190.21± 31.08	158.83± 25.40	240.94± 29.47#	263.88± 37.60#	304.89± 42.55#	251.16± 9.10#

Values are mean±SEM; n=6 in each group. All means do not show any statistically significant difference (**: p<0.001, *: p<0.05, #: p>0.05 statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

Table 9: Effect of drugs on creatinine.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 day	2.72± 0.45	1.89± 0.72	2.76± 0.82	2.81± 0.82	2.41± 0.67	2.94± 0.58	2.58± 0.86	2.69± 0.90
21st day	2.75± 0.64	3.03± 0.78*	3.84± 0.51	3.80± 0.73	3.51± 0.53	3.59± 0.45	3.63± 0.88	4.07± 0.94
42nd day	2.2± 1.10	1.52± 0.87	2.09± 1.27	2.17± 1.09*	1.69± 0.79#	1.87± 0.78#	1.92± 0.80#	1.19± 0.54*

Values are mean±SEM; n=6 in each group. All means are statistically significantly different (**: p<0.001, *: p<0.05, #: p>0.05 statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

Table 10: Effect of drugs on blood urea.

Week	Control	HFD	HFD+A	HFD+T	HFD+GC	HFD+T+GC (500 mg/kg)	HFD+T+GC (1000 mg/kg)	HFD+T+GC (2000 mg/kg)
0 day	50.05± 8.94	44.23± 5.14	47.09± 7.64	45.03± 8.37	46.11± 7.58	52.97± 7.36	48.31± 7.83	52.76± 6.44
21st day	50.60± 7.26	64.55+ 2.15*	50.63± 8.24	44.64± 8.32	48.76± 7.70	58.17± 8.51	53.64± 9.54	63.97± 7.76
42nd day	48.31± 4.85	39.51± 8.62	44.26± 8.90	31.37+ 1.09*	44.25± 8.31#	51.43± 10.88#	44.19± 9.28#	34.90± 1.94*

Values are mean ±SEM; n=6 in each group. All means are statistically significantly different (**: p < 0.001, *: p<0.05, #: p>0.05 statistically insignificant); A: Atorvastatin; T: Triphala; GC: *G. cambogia*.

The data indicated that after induction of obesity, the level of bilirubin decreased after 21days, where it was found to be 167.75±42.93 mg/dl which is higher than the control value of 210.5±48.06 mg/dl. With the co-treatment of individual standard herbal extract, triphala and *G. cambogia* extracts bilirubin value was found to be 158.83±25.40 mg/dl and 240.94±29.47 mg/dl respectively which are slightly decreased. Co-treatment of the combination of triphala and *G. cambogia*, bilirubin value was found to be 251.16±9.10 mg/dl, which is further lower.

Kidney function test (KFT)

Nephrotoxicity is one of the most common kidney problems and occurs when the body is exposed to a drug or toxin that causes damage to the kidneys. Creatinine is an important diagnostic tool in kidney diseases. Any changes in levels of creatinine in the blood are related to excretion and therefore reflect kidney function.

In our study we investigated the effects of triphala extract, *G. cambogia* extract and combination of both extracts on creatinine concentration after the HFD

induced obesity in male Wistar rats during 42 days of the experimental period. The data indicated that after induction of obesity by HFD in male Wistar rats, the level of creatinine got increased after 21days, where the value was found to be 3.84±0.51 mg/dl which is higher than the control value of 2.72±0.45 mg/dl ($p<0.001$). With co-treatment of individual, triphala extract and *G. cambogia* extract, the creatinine value was found to be 2.17±1.09 mg/dl and 1.69±0.79 mg/dl respectively which is slightly decreased. With co-treatment of the combination of triphala and *G. cambogia* the creatinine value was found to be 1.19±0.54 mg/dl, which is further less as compared to individual treatment values ($p<0.001$).

Blood urea nitrogen is an important diagnostic tool for nephrotoxicity. In our study, the blood urea level was found to be elevated in HFD induced obesity in albino Wistar rats in 21 days, in which the value increased from the control value of 50.05±8.94 to 64.55+2.15 mg/dl. After the 42 days, the treated Triphala and *G. cambogia* groups showed a slightly decreased level of blood urea where the value was found to be, 44.26±8.90 mg/dl, 31.37+1.09 mg/dl and 44.25±8.31 mg/dl respectively which is more or less equal to the control group 50.05±8.94 mg/dl ($p<0.05$).

On the other hand, co-treatment with the combination of triphala and *G. cambogia* extracts showed further decreased level of blood urea level where the value was found to be 34.90±1.94 mg/dl ($p<0.05$).

As mentioned above the determination of blood urea in rats treated with 2000 mg/kg/body weight with combination treatment showed a significant drop down towards the control value indicating protective action of combination treatment.

DISCUSSION

Our study assessed the preventive as well as the curative aspects of the herbal extracts together or individually in HFD induced rat model for obesity. The LFT and KFT related markers were assessed. Many research reports show that triphala and *G. cambogia* were effective against obesity but these results have never been investigated scientifically. Further, the effect of combination of triphala and *G. cambogia* has never been tested for obesity. As per Ayurvedic texts, triphala can dissolve accumulated fat within the body. It has been reported that treatment with triphala lead to reduced appetite and weight loss.[31] Triphala and *G. cambogia* can reduce food consumption in humans and rodent models of obesity, possibly by diverting carbohydrates and fatty acids that would have become fat in the liver, into hepatic glycogen.[32,33] This metabolic change may send signal to the brain resulting in a reduced appetite. The active component of *G. cambogia* is hydroxycitric acid (HCA), a compound that inhibits the enzyme ATP-citrate lyase, which is involved in endogenous lipid biosynthesis. Additionally, there is an increased production of hepatic glycogen in the presence of HCA, which may activate glucoreceptors leading to a sensation of fullness and reduced appetite.[28-30] Phytochemical analysis of triphala shows the presence of polyphenols, tannins, flavanoids and glycosides.[34] Tannins with a gallate group have been reported to bring about various physiological functions such as anti-lipidemic action in rats with hypercholesterolemia. Many reports have suggested functional phenolic compounds are also responsible for lipid-lowering action, beside antidiabetic and hypotriglyceridemic effects. In our study, the herbal treatment was also found to statistically improve liver function as assessed by the activity of liver-specific enzymes ALT, AST, ALP, albumin and bilirubin decrease in plasma as compared with the HFD group. The herbal treatment also decreased kidney function related parameters such as creatinine and urea as compared with the HFD group.

CONCLUSION

The herbal treatment significantly improved the clinical parameters such as ALT, AST, ALP, Albumin and Bilirubin decreased in plasma as compared with the HFD group. The herbal treatment also decreased kidney function related parameters such as creatinine and urea as compared with the HFD group. We hereby report for the first time in-vivo antiobesogenic effects of combination of triphala and *G. cambogia*.

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