Journal of Ayurvedic and Herbal Medicine

Research Article

J. Ayu. Herb. Med.
2016; 2(4): 131-135
July- August

www.ayurvedjournal.com

Evaluation of *Curcuma longa* Linn. for hypolipidaemic effects

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ABSTRACT

Ethanolic extracts of *Curcuma longa* Linn. (CLL) was evaluated for the hypolipidaemic effects in atherogenic diet fed rabbits and compared with a standard hyoplipidaemic drug lovastatin. Animals under atherogenic diet only, showed significantly raised serum cholesterol, triglycerides, VLDL and LDL levels at the 4th and 16th week of study. There was significant decrease in HDL level at 16th week of study. CLL administration at doses of 1.6 mg/kg/day and 3.2 mg/kg/day along with atherogenic diet significantly decreased all the lipid parameters except HDL at 4th and 16th week. The levels were 38.8 ± 3.06, 39.3 ± 2.16, 7.86 ± 0.43 and 9.3 ± 3.76 respectively for 1.6 mg/kg/day dose and 38.5 ± 2.25, 41.6 ± 3.32, 8.33 ± 0.66 and 6.33 ± 1.58 for 3.2 mg/kg/day dose respectively at 4th week.At 16 weeks, the lipid levels were found to be 37.6 ± 2.54, 37.6 ± 2.06, 7.53 ± 0.41 and 6.46 ± 3.06 respectively at 1.6 mg/kg/day dose and 37.5 ± 1.87, 37.5 ± 1.87, 7.5 ± 0.37 and7.16 ± 2.13 respectively at 3.2 mg/kg/day dose of CLL. Lovastatin also reduced all the above lipid parameters except HDL at 4th week and 16th week. The HDL levels in all the groups of animals under CLL as well as lovastatin increased in 4th week and it was significantly increased at 16th week. The reduction in lipid parameters of both the test doses of CLL was comparable with that of lovastatin.

Keywords: *Curcuma longa* Linn, Ethanolic extracts, Hypolipidaemic effects, Animal study.

INTRODUCTION

Lipids are one of the most important bio-molecules in human body. Cholesterol is an essential component of the human cell membrane and a precursor for steroid hormones and bile acids. Triglycerides also play an important role in transferring energy from food into body cells. However, elevation of different forms of lipids in the bloodstream, a condition generally termed hyperlipidemia, causes a constant health problem. It is always a threat to coronary arteries and the most important risk factor for coronary heart diseases because of the fact that lipids are carried in the bloodstream.

Human diet contains a variety of spices and they not only enhance the taste and flavour of food, but also exhibit a wide range of physiological and pharmacological properties. *Curcuma longa* Linn. (CLL) or turmeric is a widely used dietary spice and it is also a major ingredient of a variety of spice mix and curry powder in this part of the world. It has been demonstrated for its beneficial pharmacological effects including Alzheimer's disease[1], antibacterial[2], anti-inflammatory effects,[3] antioxidant[4] and antiplatelet[5] aggregator effects. The constituents of turmeric demethoxycurcumin, bisdemethoxycurcumin, and acetylcurcumin appear to inhibit peroxidation in rat tissues and liver microsomes.[6] Turmeric also contains sesquiterpenoids and the constituent ar-tumerone.[7] Other constituents include sugars, resins, proteins, vitamins, and minerals (including iron and potassium). Studies have been documented on the Cholesterol lowering effects of curcumin (diferuloylmethane),[8] the active principle of turmeric in hypercholesterolemic animals, probably by enhancing the activity of hepatic cholesterol-7a-hydroxylase and increasing cholesterol catabolism.[9]

The present study was undertaken to evaluate the lipid lowering effects of two doses of the ethanolic extracts of CLL in rabbits under atherogenic diet and compared with the standard drug Lovastatin.

MATERIALS AND METHOD

Present study was conducted in the Department of Pharmacology, Gauhati Medical College and after obtaining approval from the Institutional Ethics Committee (IEC), Gauhati Medical College, Guwahati, India and lipid parameters were estimated in the Department of Biochemistry, Gauhati Medical College, Guwahati.

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Preparation of extract

250 gm of dried rhizomes of CLL (local variety) was collected from local market from Guwahati. Rhizomes were cleaned thoroughly by mechanical means to remove stones, dust, lumps of soil and other foreign particles.

200 gm of the clean rhizomes were taken and ground properly to make powder. Alcohol in the strength of 85% was added to it in sufficient quantity to evenly and distinctly damp the powder. It was allowed to stand for 15 minutes and then transferred to a percolator. The ground powder was stuffed in the percolator firmly and enough of the menstrum was poured on it for saturation and the top of the percolator was closed. Percolation was done after 24 hours of maceration. This procedure was repeated for the second time by adding fresh alcohol (85%) to the previously used turmeric powder. The alcoholic solution was collected in flat bottomed (F.B.) flask. The extract was then evaporated using constant temperature until all the alcohol part evaporated leaving dry residue at the bottom of the flask.

The residue obtained after flask evaporation was mixed with the vehicle 40% w/v aqueous solution. This suspension was used as test drug doses of 1.6 mg/kg and 3.2 mg/kg body weight.[10]

Animals Treatment

The present study was carried out on 30 healthy adult rabbits (*Oryctolaguscuniculus*) of weight between 1 kg to 2 kg (Average 1.5 kg). Experimental animals were housed in the animal house under natural photoperiod at 25 ± 1^0C temperature in well ventilated condition. They were given standard diet and water ad libitum. The Bengal gram was restricted to all animals considering its hypocholesteremic properties.

They were assigned in 5 different groups containing 6 rabbits in each group as given in Table - 1.

Table 1: Grouping of animals and drug schedule

S. No.	Group	Diet and Drugs	Dose	Route
1	A	Fed Standard diet (SD)	SD = 100 gm/day/Rabbit; Vehicle = 0.2 ml distilled water	Oral
2	B	Fed Atherogenic diet (AD)	2% cholesterol + 3% coconut oil in SD	Oral
3	C	AD + Lovastatin	AD + 1.4 mg Lovastatin/rabbit (1.5 kg)	Oral
4	D	AD + CLL	AD + 1.6 mg/kg/rabbit	Oral
5	E	AD + CLL	AD + 3.2 mg/kg/rabbit	Oral

Atherogenic diet was prepared by adding 2% cholesterol and 3% coconut oil to standard diet (R.H.A. Becker, Linz M Nordolander 1991)[11].Lovastatin was used as a drug for comparison considering its well established cholesterol lowering effects[12]. The daily dose was calculated by extrapolation method (Ghosh M.N, Fundamentals of Experimental Pharmacology) based on the surface area ratio of 70 kg man to a 1.5 Kg rabbit considering the adult human dose to be 20 mg/day for adult human of 70 Kg body weight. The dose calculated out was 20 × 0.07 = 1.4 mg/kg/day (0.93 mg/Kg/day).

Blood samples were collected from the marginal ear veins of the animals after overnight fasting at the 0, 4^{th} and 16^{th} week of administration of the atherogenic diet and the study drugs. During collection of blood, care was taken to prevent haemolysis of blood. Amount of blood collected from each animal was around 1.5 ml and was kept in a sterile empty vial (SEV).

Lipid parameters

The total cholesterol was estimated using cholesterol reagent kit of Dade Behring. This method is based on the principle described first by Stadman (Stadman TC, 1957)[13] and later adapted by other workers (Flegg. H.M, 1973, Roschalauet. al., 1974)[14].Cholesterol esterase (CE) catalyzes the hydrolysis of cholesterol to produce free cholesterol which along with pre-existing free cholesterol is oxidized in a reaction catalyzed by cholesterol oxidase (CO) to form cholest-ene-3-one and hydrogen peroxide (H_2O_2). In presence of horseradish peroxidase (HPO), the hydroperoxide thus formed is used to oxidize N, N-diethyl aniline HCL-4-aminoantipyrine (DEA-HCl-AAP) to produce a chromophore that absorbs at 540 nm. The absorbance due to oxidized DEA-HCl-AAP is directly proportional to the total cholesterol concentration and is measured using polychromatic (540, 452, 700 nm) end point technique.

Triglycerides (TG) were estimated using triglyceride reagent kit of Dade Behring. It is an in-vitro diagnostic test for the quantitative determination of TG in serum of plasma.

High Density Lipoprotein (HDL) was estimated using automated HDL cholesterol Reagent Kit of Dade Behring. It is an in-vitro diagnostic test intended to quantitatively measure HDL in serum and plasma.

Low density lipoprotein (LDL) was calculated by using Friedewald's formula [15].

$$\text{LDL} = \text{Total Cholesterol} - \text{HDL Cholesterol} + \text{TG} \div 5$$

Very Low Density Lipoprotein (VLDL) cholesterol was also calculated by using the formula VLDL = TG/5.

RESULTS AND DISCUSSION

Toxicity study

The ethanolic extract of CLL was found to be safe in the doses used. No mortality was observed between 0 – 24 hours during feeding of an increasing oral dose upto 3000 mg/kg

Lipid Profile Study

Lipid profile of the different groups of animals under study were compared by using one wayAnalysis of Variance (ANOVA) test followed by Dunnet multiple comparison test and for comparison between 2 (two) groups, students t-test was used. For all tests $p < 0.05$ was used for testing significance level.

Results obtained in our study can be summarized as follows:

At 0 (zero) week: The plasma lipid profile of all the study groups of animals is presented in Table – 2.

Table 2: Plasma lipid profile in study animals at 0 week.

Groups	Cholesterol	Triglyceride	VLDL	HDL	LDL
A	34.5 ± 3.50	41.8 ± 4.62	8.3 ± 0.92	8.61 ± 2.63	17.9 ± 4.02
B	41.6 ± 4.59	52.0 ± 2.52	10.4 ± 0.50	22.16 ± 4.57	9.1 ± 3.91
C	43.3 ± 2.58	50.1 ± 3.81	10.03 ± 0.76	15.5 ± 3.88	17.8 ± 5.97
D	41.6 ± 3.93	49.6 ± 2.16	9.93 ± 0.42	14.83 ± 3.12	16.9 ± 3.19
E	38.0 ± 2.44	49.0 ± 6.48	9.8 ±1.36	16.0 ± 4.19	12.2 ± 6.02

The results show that the mean total cholesterol level in normal animals, i.e. Gr. A varied from 30 – 48 mg/dl with a mean value of 34.5 ± 3.50. The levels for the Gr. B, Gr. C, Gr. D and Gr. E were 41.6 ± 4.59, 43.3 ± 2.58, 41.6 ± 3.93 and 38.0 ± 2.44respectively. The TG levels for the above groups were 41.8 ± 4.62, 52.0 ± 2.52, 50.1 ± 3.81, 49.6 ± 2.16 and 49.0 ± 6.48 respectively. Similarly the estimated mean values of VLDL for the above groups were 8.3 ± 0.92, 10.4 ± 0.50, 10.03 ± 0.76, 9.93 ± 0.42 and 9.8 ± 1.36respectively. The calculated mean ± SD values of HDL for these 5 (five) groups of animals were 8.61 ± 2.63, 22.16 ± 4.57, 15.5 ± 3.88, 14.83 ± 3.12 and 16.0 ± 4.19 and the mean LDL values were 17.9 ± 4.02, 9.1 ± 3.91, 17.8 ± 5.97, 16.9 ± 3.19 and 12.2 ± 6.02 respectively.

At 4 weeks: The lipid profiles of the experimental rabbits for different groups are presented in Table – 3.

Table 3: Plasma lipid profile in study animals at 4 weeks

Groups	Cholesterol	Triglyceride	VLDL	HDL	LDL
A	41.5 ± 4.08	51.16 ± 2.92	10.23 ± 0.58	14.3 ± 2.80	16.93 ± 5.69
B	#78.5 ± 8.50	#93.3 ± 5.16	#18.6 ± 1.03	18.3 ± 2.25	#41.5 ± 9.23
C	*48.6 ± 6.28	*50.3 ± 2.06	*10.06 ± 0.41	26.8 ± 2.85	*14.03 ± 6.35
D	*38.8 ± 3.06	*39.3 ± 2.16	*7.86 ± 0.43	21.66 ± 2.42	*9.3 ± 3.76
E	*38.5 ± 2.25	*41.6 ± 3.32	*8.33 ± 0.66	22.16 ± 2.31	*6.33 ± 1.58
One WayANNOVA	df 4,25F 59.98p < 0.01	4,25261.93<0.01	4,25261.93< 0.01	4,2520.14< 0.01	4,2533.48< 0.01

*P < 0.01 as compared to group B, #P < 0.01 as compared to group A; n=6 in each group

It was observed that the atherogenic diet significantly increased the serum cholesterol level at the end of 4th week. While the the mean cholesterol level for the animals with atherogenic diet was 78.5 ± 8.50, but the animals in normal diet was 41.5 ± 4.08. Animals administered CLL @ 1.6 mg/kg/day and 3.2 mg/kg/day along with atherogenic diet showed the reduced cholesterol levels of 38.8 ± 3.06 and 38.5 ± 2.25, which are statistically significant. (Table – 3,4,5). The Triglyceride (TG), VLDL and LDL levels in normal animals were 51.16 ± 2.92, 10.23 ± 0.58 and 16.93 ± 5.69 respectively. Animals under atherogenic diet only, showed significantly raised levels of the above parameters which are recorded as 93.3 ± 5.16, 18.6 ± 1.03 and 41.5 ± 9.23 respectively when compared with normal animals. (Table-3) CLL administration to animals, even in presence of atherogenic diet, significantly reduced the levels of the above parameters when compared with the animals under atherogenic diet only. The levels of TG, VLDL and LDL were found to be 39.3 ± 2.16, 7.86 ± 0.43 and 9.3 ± 3.76 respectively for the Group D animals and they were respectively 41.6 ± 3.32, 8.33 ± 0.66 and 6.33 ± 1.58 for Group E animals. (Table – 3,4,5)

Lovastatin administration to animals along with atherogenic diet also significantly reduced the levels of total cholesterol, TG, VLDL and LDL. The recorded levels were 48.6 ± 6.28, 50.3 ± 2.06, 10.06 ± 0.41 and 14.03 ± 6.35 respectively (Table – 3).

The HDL level for the animals under normal diet was recorded as 14.3 ± 2.80 and it was 18.3 ± 2.25 in the animals under atherogenic diet. Administration of Lovastatin raised the level of HDL and recorded 26.8 ± 2.85. Administration of CLL in both doses also raised the same levels recorded as 21.66 ± 2.42 and 22.16 ± 2.31 respectively for Gr.D and Gr.E which are statistically insignificant. (Table-3)

Table 4: Comparison of Plasma lipid profile between animals with atherogenic diet only and animals with atherogenic diet plus CLL (1.6 mg/kg/day) at 4 weeks.

Group	Cholesterol	Triglyceride	VLDL	HDL	LDL
B	78.5 ± 8.5	93.3 ±5.16	18.6 ± 1.03	18.3 ± 2.25	41.5 ± 9.23
D	38.8 ± 3.06	39.3 ± 2.16	7.86 ± 0.43	21.66 ± 2.42	9.3 ± 3.76
t	10.75	23.68	23.86	2.42	7.89
df	10	10	10	10	10
p	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

n=6 in each group

Table 5: Comparison of Plasma lipid profile between animals with atherogenic diet only and animals with atherogenic diet plus CLL (3.2 mg/kg/day) at 4 weeks.

Group	Cholesterol	Triglyceride	VLDL	HDL	LDL
B	78.5 ± 8.5	93.3 ± 5.16	18.6 ± 1.03	18.3 ± 2.25	41.5 ± 9.23
E	38.5 ± 2.25	41.6 ± 3.32	8.33 ± 0.66	22.16 ± 2.31	6.33 ± 1.58
t	11.14	20.68	20.95	2.94	9.18
df	10	10	10	10	10
p	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

At 16 weeks: The lipid profiles of the experimental rabbits for different groups are presented in Table – 6.

Table 6: Plasma lipid profile in study animals at 16 weeks

Groups	Cholesterol	Triglyceride	VLDL	HDL	LDL
A	61.3 ± 3.50	78.8 ± 5.15	15.76± 1.03	24.3 ± 3.98	21.23 ± 3.53
B	#198 ± 7.01	#115.8 ± 5.60	#23.06 ± 1.20	#13.16 ± 3.13	#161.76 ± 8.15
C	*46.3 ± 2.50	*39.1 ± 2.78	*7.83 ± 0.55	*28.3 ± 3.32	*7.93 ± 3.78
D	*37.6 ± 2.54	*37.6 ± 2.06	*7.53 ± 0.41	*23.6 ± 2.87	*6.46 ± 3.06
E	*37.5 ± 1.87	*37.5 ± 1.87	*7.5 ± 0.37	*22.8 ± 1.94	*7.16 ± 2.13
One WayANNOVA	df4,25 F1870.62 p< 0.01	4,25 499.71 < 0.01	4,25 463.31 < 0.01	4,25 19.23 < 0.01	4,25 1287.72 < 0.01

*P < 0.01 as compared to group B, #P < 0.01 as compared to group A n=6 in each group

Animals fed atherogenic till 16th week had their serum cholesterol level increased significantly when compared with the animals under normal diet. While the mean cholesterol level for the animals with atherogenic diet was 198 ± 7.01, but the animals in normal diet was 61.3 ± 3.50. Animals administered 2 different doses of CLL(1.6 m/kg/day and 3.2 mg/kg/day)along with atherogenic diet showed the reduced cholesterol level of 37.6 ± 2.54 and 37.5 ± 1.87, which are statistically significant. (Table – 6,7,8).The TG, VLDL and LDL levels in normal animals were 78.8 ± 5.15, 15.76 ± 1.03 and 21.23 ± 3.53 respectively. Animals under atherogenic diet only showed significantly raised levels of the above parameters and recorded as 115.8 ± 5.60, 23.06 ± 1.20 and 161.76 ± 8.15 respectively when compared with normal animals. CLL administration to animals, even in presence of atherogenic diet, significantly reduced the levels of the above parameters when compared with the animals under atherogenic diet only. The levels were found to be 37.6 ± 2.06, 7.53 ± 0.41 and 6.46 ± 3.06 respectively at 1.6 mg/kg dose and 37.5 ± 2.06, 7.5 ± 0.37 and7.16 ± 2.13 respectively at 3.2 mg/kg of CLL. (Table – 6,7,8).

Lovastatin administration to animals along with atherogenic diet also significantly reduced the levels of total cholesterol, TG, VLDL and LDL. The recorded levels were 46.3 ± 2.50, 39.1 ± 2.78, 7.83 ± 0.55 and 7.93 ± 3.78 respectively (Table – 6).

The HDL level for the animals under normal diet was recorded as 24.3 ± 3.98 and it showed a significant reduction to 13.16 ± 3.13 in the animals under atherogenic diet. Administration of Lovastatin and CLL also significantly raised the level of HDL in their respective groups of animals and recorded as 28.3 ± 3.32 for Lovastatin group, while they are 23.6 ± 2.87 and 22.8 ± 1.94 respectively for the doses of 1.6 mg/ kg and 3.2 mg/kg of CLL. (Table – 6)

Table 7: Comparison of Plasma lipid profile between animals with atherogenic diet only and animals with atherogenic diet plus CLL (1.6 mg/kg/day) at 16 weeks.

Group	Cholesterol	Triglyceride	VLDL	HDL	LDL
B	198.5 ± 7.01	115.8 ± 5.60	23.06 ± 1.20	13.16 ± 3.13	161.76 ± 8.15
D	37.6 ± 2.54	37.6 ± 2.06	7.53 ± 0.41	23.6 ± 2.87	6.46 ± 3.06
t	52.55	32.18	30.45	6.03	43.62
df	10	10	10	10	10
p	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

n=6 in each group

Table 8: Comparison of Plasma lipid profile between animals with atherogenic diet only and animals with atherogenic diet plus CLL (3.2 mg/kg/day) at 16 weeks.

Group	Cholesterol	Triglyceride	VLDL	HDL	LDL
B	198.5 ± 7.01	115.8 ± 5.60	23.06 ± 1.20	13.16 ± 3.13	161.76 ± 8.15
E	37.5± 1.87	37.5± 1.87	7.50 ± 0.37	22.8 ± 1.94	7.16 ± 2.13
t	54.2	32.48	30.50	6.42	44.81
df	10	10	10	10	10
p	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

CONCLUSION

The present study shows that the atherogenic diet significantly raised serum cholesterol, triglycerides, VLDL and LDL at 4^{th} and 16^{th} weeks, whereas it showed a decrease in HDL at 16th week of feeding. Ethanol extracts of turmeric at the dose of 1.6 mg/kg/day and 3.2 mg/kg/day along with the atherogenic diet showed lowering of all lipid parameters, except HDL which it seemed to increase. The present study on an experimental model of rabbit re-establishes the already reported beneficial pharmacological effects of the widely and most commonly used dietary spice as well as age old traditional medicine as an agent capable of lowering serum lipids. This warrants a further study to explore its mechanism of action for potential drug development in future. Detail biochemical and molecular level studies on its effect on lipid metabolism and hepatic enzymes are required.

ACKNOWLEDGEMENTS

The authors are thankful to the authorities of Gauhati Medical College & Hospital, Guwahati, Assam India for allowing carrying out the present study.

CONFLICT OF INTEREST

There are no conflicts of interest to declare.

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HOW TO CITE THIS ARTICLE

Bordoloi P, Lahkar M. Evaluation of *Curcuma longa* Linn. for hypolipidaemic effects. J Ayu Herb Med 2016;2(4):131-135.