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Research Article

THE ANTIHYPERLIPIDAEMIC ACTIVITY OF AQUEOUS *LEUCAS URTICIFOLIA* EXTRACT IN TRITON WR-1339 INDUCED HYPERLIPIDAEMIC RATS

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Abstract

In the present study, an aqueous extract from *Leucas urticifolia* was evaluated for its antihyperlipidaemic activities using Triton WR-1339 induced hyperlipidemic rats as experimental model. Hyperlipideamia was developed by intraperitonial injection of Triton (100 mg/kg body weight). The animals were divided into control, hyperlipidaemic control, fenofibrate, and aqueous extracts (100, 200 and 400 mg/kg) of *L. urticifolia* treated groups. At 6 h after intragastric administration of *L. urticifolia* aqueous extracts (100, 200 and 400 mg/kg body weight) to triton induced hyperlipidaemic rats caused a significant decrease on their plasma TG, LDL and VLDL-cholesterol level and cause significant rise of HDL-cholesterol level, but the TC level was not altered by all three dose level. After 24 h after treatment, there was no significant change in plasma lipid parameters observed. In fenofibrate treated rats, plasma TC, TG, LDL and VLDL were lowered significantly till 24 hour. Hense the results indicating that this aqueous herb extract may contain products that lower plasma lipid concentrations and might be beneficial in treatment of hyperlipideamia and related diseases.

Key words: *Leucas urticifolia*, Triton WR-1339, Antihyperlipidaemic, Plasma lipid parameters.

Introduction

Hyperlipidemia has been ranked as one of the greatest risk factors contributing to the prevalence and severity of coronary heart diseases[1]. Hyperlipidemia is characterized by elevated serum total cholesterol (TC) and low density (LDL) and very low-density lipoprotein (VLDL) cholesterol and decreased high-density lipoprotein (HDL) levels[2].

Coronary heart disease, stroke, atherosclerosis and hyperlipidemia are the primary cause of death[3]. It is now established that hyperlipidaemia represents a major risk factor for the premature development of atherosclerosis and its cardiovascular complications[4]. Among these hypercholesterolemia and hypertriglyceridemia are closely related to ischemic heart disease[5]. A logical strategy to prevent or treat atherosclerosis and reduce the incidence of cardiovascular disease events is to target the hyperlipidaemia by diet and/or lipid-lowering drugs[6]. Currently available hypolipidemic drugs have been associated with a number of side effects[7]. The consumption of synthetic drugs leads to hyperuricemia, diarrhoea, nausea, myositis, gastric irritation, flushing, dry skin and abnormal liver function[8].

Hyperlipidemia is classified into a primary and a secondary type, which indicates the complexities associated with disease. The primary disease may be treated by anti-lipidemic drugs but the secondary type originating from diabetes, renal lipid nephrosisorhypothyroidism demands the treatment of the original disease rather than hyperlipidemia[9]. Consumption of much fat may lead to the production of extra VLDL, resulting in the formation of large amounts of LDL which may stick to the walls of the blood vessels if the quantity of HDL is insufficient, causing blockages for the normal flow of blood. Therefore, improvement in human diet is highly recommended for disease prevention[10].

In many developing countries, most hyperlipidaemic individuals use medicinal plants as folk medicine to treat hyperlipideamia and atherosclerosis because they have no side effects and is relatively cheap, locally available. They are effective in reducing the lipid levels in the system[11]. Therefore, there is a strong interest locally to search for natural hypolipemic substances derived from medicinal plants used. A vast number of these plants are to receive attention in this regard and have been shown to lower plasma lipid levels, some examples are *Gacinia cambogia*[12], *Zingiber officinale*[13] and *Emblica officinalis*[14], *Aleurites moluccana*[15].

Leucas Urticifolia belong to family Lamiaceae is an annual herb distributed in the Rajasthan, Punjab, Baluchistan, Sindh and Rajputana desert of Pakistan[16,17]. It is commonly known as kubo in gujrati[18], darkan in rajasthani[16], it is also known as Goma or Guldora[17]. The plant is tarditionaly also used for the treatment of diarrhea, dysentery, uterine haemorrhages, dropsy, gravel, cystitis, calculus, bronchial catarrh, skin diseases, fever and various types of

mental disorders. The decoction of the leaves and apical shoots with gur is used locally as an abortifacient up to 3 months of pregnancy[19].

Leucas urticifolia is reported to have Triterpenes like: Leucisterol, β-sitosterol, and ursolic acid[20], Diterpene: Momilactone-A[21], Flavonoids: Leufolins A, Leufolins B[22], Acids and esters: Urticic acid, Methoxybenzyl benzoate, 4-hydroxy benzoic acid[20]. The flavonoidal glucosides leufolins A and B of Leucas Urticaefolia exhiited significant inhibitory potential against the enzyme butyrylcholinesterase[22].

A survey of literature revealed that no systematic approach has been made to study antihyperlipidaemic activity of this plant and revealed the presence of flavonoids, which have potent antioxidant activity and helpful in lowering plasma lipids. Hence the present study was undertaken to assess activity of aqueous extract of LUAE in Triton WR-1339 induced hyperlipidaemic rats.

Material and methods:

Plant material

The leaves of *L. urticifolia* were dried in shade and powdered coarsely. For preparation of aqueous extract, 1 kg of powdered leaves was macerated with 4 liter of distilled water for seven days with intermittent stirring, filtered and concentration. The dried extract was stored at 4°C until used. The extract was subjected to preliminary phytochemical tests.

Preliminary phytochemical studies

The aqueous extract of LUAE subjected to qualitative chemical investigation for the identification of the phytoconstituents- sterols, glycosides, saponins, carbohydrates, alkaloids, flavonoids, tannins, proteins by their standard qualitative tests[23].

Drug formulations

All the preparations to be given by oral route were prepared freshly in distilled water just before dosing.

Animals

Healthy Wistar albino rats of either sex and of approximately same age (12 to13 weeks), weighing between 150-200 g were used for the study. The animals were acclimatized by keeping them in animal house facility of Sri Balaji College of Pharmacy, Jaipur, Rajasthan. They were housed individually in polypropylene (32x24x16 cm)

cages containing bedding material as husk and maintained under controlled conditions of temperature (23±2^{o}c), humidity (55±5%) and 12 h light and 12 h dark cycles, and were fed with commercial pellet rat chow and water ad libitum.

The norms for Good Laboratory Practice were followed for care of laboratory animals. The studies were conducted after obtaining the approval from Institutional Animal Ethical Committee clearance of Sri Balaji College of Pharmacy, Jaipur, Rajasthan. The animal house facility of this division is approved by Govt. of India under the Ministry of Environment and Forest (Reg no. 1212/ac/08/CPCSEA). The experimental protocol was approved by Institutional Animal Ethical Committee (IAEC) of Name of College and Place (Letter No. SBCP/IAEC/10-465) with CPCSEA Reg. no. 1212/ac/08/CPCSEA).

Acute oral toxicity study

Acute oral toxicity study for the test extract (LUAE) was carried out using OCED guideline 425 (modified, adopted 23rd march 2006)[24].

Antihyperlipidaemic activity in triton induced hyperlipidemia

For this study the overnight fasted rats were divided into six groups of six animals each. The first group served as normal control (Control), received intraperitoneal administration of normal saline; the second, hyperlipidaemic group (Triton Control), was treated with intraperitoneal injection of Triton WR-1339 at a dose of 100 mg/kg in normal saline; the animals of third group (Fenofibrate) were treated with intragastric administration of fenofibrate (20 mg/kg). The fourth group (LUAE-100) receives LUAE at the dose of 100 mg/kg; the fifth group (LUAE-200) received 200 mg/kg of LUAE by gavage; the animals of sixth group (LUAE-400) received 400 mg/kg of LUAE. Except normal control group all the animals will be treated with intraperitoneal injection of Triton WR-1339 at a dose of 100 mg/kg body weight. Till the 24 h of study, animals had access only to water[25-26].

Blood collection and biochemical analysis

Before the treatment (0 h) and after 6, 24 and 48 h from treatments, animals were anaesthetized with diethyl ether and blood was taken from their tail vein using heparinized capillary. The blood samples were immediately centrifuged (4500 rpm/10 min) and plasma used for lipid analysis such as plasma total cholesterol (TC), triglycerides (TG) and HDL-cholesterol were quantified using enzymatic kits (Agapee diagnostics). The LDL-

cholesterol and VLDL-cholesterol were calculated by using Friedwald's (LDL=Total cholesterol-[TG/5]-HDL)[27] and Norbert formulas (VLDL=TG/5)[28].

Statistical analysis

Results are expressed as mean ± S.D. Statistical differences between means were determined by One-way ANOVA followed by Tukey's post hoc test using GraphPad Prism 5. P value <0.05 was considered significant.

Results

Phytochemical screening

The phytochemical tests revealed that the aqueous extract of *L. urticifolia* leaves possess alkaloids, flavonoids, tannins, glycosides, carbohydrates and proteins in aqueous extracts.

Acute toxicity study

The alcoholic extract of *L. urticifolia* was found to be safe upto dose of 2000 mg/kg b.w. without produce any mortality and other toxic effects. Hence the $1/20^{th}$, $1/10^{th}$, and $1/5^{th}$ of doses was taken, which were found to be 100, 200 and 400 mg/kg body weight.

Antihyperlipidaemic activity

The effect of LUAE on levels of plasma lipid parameters after triton induced hyperlipidaemia is reported in Tables 1. In comparison with control group, Triton WR-1339 caused a marked increase in TG, LDL, VLDL and caused marked fall in HDL-cholesterol plasma concentrations measured on 6 and 24 h after injection. But the plasma concentration level of TC was not increased significantly by the triton till the completion of study. After 48 hours of Triton WR-1339 injection the values of all lipid parameters came back to normal level.

After 6 h treatment, the administration of 100, 200 and 400 mg/kg doses of alcoholic *L. urticifolia* extract in Triton injected rats, the plasma TG, LDL and VLDL were decreased ($P<0.05$), with respect to triton control group. HDL-cholesterol was increased ($P<0.01$). At 24 and 48 h after treatment, LUAE at all three dose levels (100, 200 and 400 mg/kg) didn't show any significant change in plasma lipid parameters. The levels of plasma lipid parameters at 48 h after treatment found to be comparable with normal control group level. The fenofibrate significantly reduced the level of TC, TG, VLDL and LDL-cholesterol levels ($P<0.05$) and cause increase in HDL-cholesterol

level significantly (P<0.05) compared to triton control group, till 24 h after treatment. The levels of all plasma lipid parameters reached to their normal values when compared to control group.

Discussion

Triton WR-1339 has been widely used to block clearance of triglyceride-rich lipoproteins to induce acute hyperlipidaemia in several animals[29]. This model is widely used in rats and used for screening of hypolipidaemic drugs[30] because it is less time consuming and easy to handle. Hence, many plants such as *Vaccinum myrtillus*[31] and *Phyllanthus niruri*[32] have been investigated for their acute hypolipidaemic activity in Triton WR-1339 induced hyperlipidaemic animals. A parenteral administration of a dose of Triton WR-1339 to adult rats induced hyperlipidaemia. The maximum plasma triglycerides was reached at 20 h, followed by a decline to normal values[30,32,33]. In our hand, the same model gave similar pattern of lipid profile changes either at 7 or 24 h after Triton injection (Tables 1) and demonstrates the feasibility of using it of acute hyperlipidaemia, to assess the antihyperlipidaemic activity of LUAE.

Table-1: Effect of *Leucas urticifolia* on plasma TC, TG, HDL, VLDL and LDL levels in Triton WR-1339 induced hyperlipidaemia.

Groups		Control	Triton	Fenofibrate	LUAE-100	LUAE-200	LUAE-400
TC	0 Hr.	98.60±3.12	98.94±1.95	97.60±1.72	99.40±1.15	98.87±1.99	100.44±2.55
	6 Hr.	100.00±4.75	104.77±2.54^{a0}	78.27±2.97^{a3,b3}	100.60±3.89^{a0,b0}	99.27±3.24^{a0,b0}	100.27±5.13^{a0,b0}
	24 Hr.	99.77±2.81	102.94±3.00^{a0}	83.44±1.78^{a3,b3}	104.60±2.91^{a0,b0}	100.74±2.03^{a0,b0}	99.77±1.81^{a0,b0}
	48 Hr.	100.10±3.58	99.77±2.53^{a0}	92.77±6.28^{a1,b1}	97.94±2.39^{a0,b0}	98.60±3.43^{a0,b0}	96.60±3.79^{a0,b0}
TG	0 Hr.	109.21±6.46	109.04±5.33	106.71±7.59	109.71±5.93	109.71±3.20	111.04±9.47
	6 Hr.	104.71±7.05	152.58±6.57^{a3}	72.38±3.45^{a3,b3}	141.21±6.74^{a3,b1,c3}	135.88±4.79^{a3,b3,c3}	130.71±5.33^{a3,b3,c3}
	24 Hr.	104.38±4.85	127.04±3.35^{a3}	85.71±4.90^{a3,b3}	123.38±7.49^{a3,b0,c3}	120.04±8.89^{a2,b0,c3}	120.38±8.76^{a3,b0,c3}
	48 Hr.	103.88±7.74	112.88±5.54^{a0}	96.21±3.15^{a0,b3}	111.21±5.07^{a0,b0,c3}	110.21±4.53^{a0,b0,c3}	108.71±5.73^{a0,b0,c2}
HDL	0 Hr.	58.79±2.87	58.29±1.99	57.96±1.68	59.12±2.43	58.62±2.61	59.96±3.15
	6 Hr.	59.12±3.14	39.29±3.21^{a3}	59.96±2.47^{a0,b3}	47.62±2.41^{a3,b2,c3}	50.96±4.51^{a2,b3,c2}	51.62±5.67^{a1,b3,c2}
	24 Hr.	58.46±2.74	50.62±3.64^{a3}	60.12±1.98^{a0,b3}	52.29±3.15^{a2,b0,c3}	52.96±3.20^{a1,b0,c3}	54.62±2.27^{a0,b0,c1}
	48 Hr.	58.29±1.98	56.62±2.59^{a0}	59.79±2.37^{a0,b0}	57.29±2.01^{a0,b0}	57.96±3.43^{a0,b0}	57.62±3.44^{a0,b0}
LDL	0 Hr.	17.96±1.06	18.83±1.29	18.30±1.51	18.33±1.18	18.30±0.64	18.26±1.89
	6 Hr.	19.93±1.41	34.96±1.31^{a3}	3.83±0.69^{a3,b3}	24.73±1.34^{a3,b1,c3}	21.13±0.95^{a3,b3,c3}	22.50±1.06^{a3,b3,c3}
	24 Hr.	20.43±0.97	26.90±0.67^{a3}	6.16±0.98^{a3,b3}	27.63±1.49^{a3,b0,c3}	23.76±1.77^{a2,b0,c3}	25.18±1.75^{a3,b0,c3}

	48 Hr.	21.03±1.54	20.56±1.10^{a0}	13.73±0.62^{a3,b3}	18.40±1.01^{a0,b0,c3}	18.60±0.90^{a0,b0,c3}	17.23±1.14^{a0,b0,c2}
VLDL	**0 Hr.**	21.84±1.28	21.80±1.38	21.34±1.98	21.94±1.88	21.94±1.86	22.20±1.88
	6 Hr.	20.94±3.36	30.51±1.69^{a3}	14.47±0.41^{a3,b3}	28.24±2.57^{a3,b1,c3}	27.17±1.96^{a3,b3,c3}	26.14±0.73^{a3,b3,c3}
	24 Hr.	20.87±1.98	25.40±1.62^{a3}	17.14±0.87^{a3,b3}	24.67±1.94^{a3,b0,c3}	24.00±2.82^{a2,b0,c3}	24.07±2.59^{a3,b0,c3}
	48 Hr.	20.77±1.54	22.57±1.10^{a0}	19.24±0.623^{a0}	22.24±1.01^{a0}	22.04±0.90^{a0}	21.74±1.14^{a0}

Values are expressed as mean ± SD on six animals in each group; 'a0' no significant, 'a1' P<0.05, 'a2' P<0.01, 'a3' P<0.001 when compared to control group; 'b0' no significant, 'b1' P<0.05, 'b2' P< 0.01, 'b3' P<0.001 when compared to triton control group; 'c0' no significant, 'c1' P<0.05, 'c2' P<0.01, 'c3' P<0.001 when compared to standard simvastatin treated group.

Present results clearly show LUAE at a dose of 100, 200 and 400 mg/kg body weight significantly lowered both plasma triglycerides, LDL and VLDL-cholesterol levels at 6 h and 24 h (Table 1). The large increase in plasma triglycerides due to Triton WR-1339 injection results mostly from an increase of VLDL secretion by the liver accompanied by a strong reduction of VLDL and LDL catabolism[34]. The reduction of total cholesterol by the LUAE was associated with a decrease of its LDL fraction, which is the target of several hypolipidemic drugs. This result suggests that cholesterol-lowering activity of the herb extract can be result from the rapid catabolism of LDL cholesterol through its hepatic receptors for final elimination in the form of bile acids as demonstrated by Khanna et. al.[32].

Flavonoids exhibited a variety of pharmacological activites, including the anti-atherogenesis and antioxidant effect[35]. Furthermore, quantification of tannins and flavonoids contents in plant samples confirmed by preliminary phytochemical tests. The results strongly suggests that the hypolipidemic activity of this medicinal plant could be attributed to the presence of the valuable polyphenolic compounds. Increase in plasma lipid, cholesterol and triglycerides levels is related to significant changs in lipid metabolism and structure[36].

Abnormalities in cellular cholesterol metabolism could partly be responsible for the changes in the plasma cholesterol levels in diabetes[37]. Diabetes is also associated with hyperlipidemia. Serum triglycerides have been decreased significantly in diabetic rats after extract supplementation. These effects may be due to low activity of cholesterol biosynthesis enzymes and / or low-level lipolysis. This extract supplementation also resulted in significant attenuation in the level of LDL and HDL in serum towards the control level, which again strengthens the hypolipidemic effect of this extract. Thus, our study showed that administration of aqueous extract of 400 mg kg-1 of Camellia sinensis was more effective to manage hyperlipidemia. The active ingredients present here may recover the disorders in lipid metabolism noted in hyperlipidemic state.

In conclusion, our results apparently show the antihyperlipidaemic action of aqueous *Leucas urticifolia* extract in triton induced hyperlipidaemic rats. This is the first study which investigates the hypolipidaemic activity of aqueous *L. urticifolia* extract and the results found are encouraging for further assessment to elucidate both mechanisms, and hypolipidaemic action on chronic hyperlipidaemia models and to identify the bioactive compounds.

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