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Antihyperlipidemic activity of flower extract of *Tecoma stans* against dexamethasone-induced hyperlipidemia in rats

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ABSTRACT

Introduction

Natural products derived from plants kingdom play a vital part in preventing or treating various diseases or disorders in humans. Hyperlipidemia is one of the major pathological factors of cardiovascular diseases and diabetes mellitus. On the other hand, *Tecoma stans* belonging to the family Bignoniaceae, was found to possess many pharmacological activities such as anti-inflammatory, antibacterial, and anti cancer activities along with CNS depressant activities.

Materials and Methods

The present study is an attempt to investigate its antihyperlipidemic activity by in vivo animal model. Hyperlipidemia model can be induced by administered with dexamethasone in rats with significant increase in serum cholesterol and triglyceride (TG) levels along with increase in the atherogenic index.

Results

The ethanolic extract of flowers of *T.stans* (200 and 400 mg/kg) treatment has shown significant inhibition against dexamethasone-induced hyperlipidemia in rats by maintaining the serum levels of cholesterol, TGs and near to the normal levels.

Keywords: Tecoma Stans, Dexamethasone, Hyperlipidemia, Cholesterol and Triglyceride

INTRODUCTION

Around 80% of the world population uses the herbal medicines for primary health care mostly in the developing countries. [1] Because of their safety, efficacy, cultural acceptability, better compatibility with the human body and lesser side effects, they stood still. There was mention about the usage of herbal medicines for age-related diseases such as memory loss, diabetic wounds, osteoporosis, and immune and liver disorders in various ancient literatures, for which no modern medicine or only palliative therapy is available.

Some of the life-saving and essential drugs discovered from medicinal plants such as digoxin, morphine, emetine, aspirin, and ephedrine were known to modern therapeutics several centuries ago. There was a statement described by Namdeo about secondary metabolites derived from plants; he stated that about a 1/4th of all suggested pharmaceuticals in developed countries containing compounds that are directly or indirectly derived from plants. [2] There is a belief that green medicine is safe and trustworthy.

Today, there is a widespread curiosity in drugs derived from plants. At present, many pharmaceutical companies are concentrating extensive research on plant materials for their potential medicinal value. [3] As per the World Health Organization, 4 billion people (80%) of the world population are using plant-derived products as medicine for some aspect of primary health care. Out of 119 plant-derived medicines, approximately 74% are used nowadays that are directly correlated with their traditional practice as plant medicines by native cultures.Atherosclerosis is one of the leading causes of death in the world both in developed countries and as well as developing countries like India. [4] The elevated levels of low-density lipoprotein (LDL) and very LDLs (VLDLs) associated with cholesterol and triglycerides (TGs) is one of the primary risk factors for atherosclerosis. By targeting the atherogenic process, we can treat hyperlipidemia is one of the palliative treatment approaches for atherosclerosis. [5] A wide number of allopathic antihypolipidemics are available in the market, but they were not popularized due to their side effects and contraindications. To overcome that recently herbal hypolipidemics have gained importance to fill the voids. [6],

Tecoma stans was used as a folklore medicine for numerous ailments. The flowers of the *T.stans* were used medicinally for reducing pain and swelling. Recent pharmacological studies have shown Anti cancer activity [7, 8], anti-nociceptive and anti-inflammatory activity [9], CNS depressant activity [10], Anti-obesity and Hypolipidemic activity [11], Hepatoprotective activity [12], faecal dropping activity [13], Antiurolithiatic Activity [14], Ameliorative activity [15], Anti Oxidative damage activity [16], Antidepressant activity [17], Diuretic activity [18], wound healing activity [19], anti microbial activity [20]. Their effects against Dexamethasone-induced Hyperlipidemia were not reported elsewhere. The present investigation was to carry out the antihyperlipidemic effect of ethanolic extract of *T.stans* (EETS) against dexamethasone-induced hyperlipidemia in rats.

MATERIALS AND METHODS

Chemicals

The solvents used for the study (petroleum ether and ethanol) were obtained from SD Fine Chemicals India Ltd. and were of laboratory grade. Gemfibrozil was purchased from Medindia, dexamethasone and all other chemicals used in the present study were purchased from Merck, India and of analytical grade.

Plant materials

T.stans plant flowers were collected in the month of August 2019 from Namakkal, Tamilnadu, India. The plant was then taxonomically identified and authenticated by the botanist in the botanical surveyor of India, Coimbatore.

Experimental animals

Male Wister rats (150-180 g) of approximately the same age, obtained from Elite labs, Hyderabad, India, were used for the study. They were kept in polypropylene animal cages and fed with normal rodent pellet diet (Hindustan Lever Limited, Hyderabad) and water *ad libitum*. All the animals were exposed to an alternate cycle of 12 h of darkness and 12 h of light. Before each test, the animals were withdrawn from food for at least 12 h. All the experiments were carried out in the morning as per the current guidelines for care of laboratory animals and the ethical guidelines for investigations of experimental pain in conscious animals. The standard oral gastric cannula and syringe were used for drug administration in experimental animals. [21]

Methodology

Extraction

The plant flowers were dried in shade, after confirmation of the moisture content limits, the dried flowers were coarsely powdered using a mechanical grinder. Then, the powder was passed through sieve No. 40 and stored in an airtight container for the extraction.

Preparation of extracts [22]

Petroleum ether extract of T.stans

The powder of the dried flowers of *T.stans* was extracted with petroleum ether at temperature 60-80°C up to 48 h. After completion of extraction, the solvent was filtered, and the powder was separated. The crude extract was subjected to distillation and solvent was removed. Dark green-colored residue was obtained, and it was stored in desiccators.

Ethanol extract of T.stans

The marc left after petroleum ether extraction was dried and then extracted with 95% ethanol at 75-78°C up to 48 h. After completion of extraction, the solvent was distilled off from the crude extract. Dark brown-colored residue was obtained. The residue was concentrated and then stored in desiccators.

Aqueous extract of T.stans

The marc left after ethanol extraction was dried and then macerated with distilled water, up to 48 h. After completion of extraction, it was filtered and the solvent was removed by evaporation to dryness on a water bath. Brown color residue was obtained, and it was stored in desiccators.

Pharmacological evaluation

Dexamethasone-induced hyperlipidemia model

Glucocorticoid hormonal level elevation induces the plasma lipid concentration but varies from species to species. Few synthesis of triacylglycerol in the liver is stimulated by the injection of glucocorticoid in rats and consequently may lead to the accumulation of fatty liver. The stimulation of the TG production could lead to increased secretion of VLDL. Increasing VLDL secretion has been reported when dexamethasone is injected for several days in rats. The increase in TG level induces imbalance in lipid metabolism leads to hyperlipidemia. Similarly, dexamethasone treatment in newborn rats for 4 days showed widespread increase in serum lipids. [23-26]

Dexamethasone-induced hyperlipidemia in rats

Induction of hyperlipidemia

Hyperlipidemia will be induced using dexamethasone; a glucocorticoid is known to evoke plasma lipid elevation. Dexamethasone (10 mg/kg/day, subcutaneous) was administered to rats for 8 days to induce hyperlipidemia. The animals were divided into five groups each group contains six rats details were shown in Table 3.

Procedure

All the animals in Groups II, III, IV, and V were subjected to subcutaneous injection of dexamethasone (10 mg/kg/day, S.C) for 8 days to induced hyperlipidemia. The animals in normal and hyperlipidemic control groups were received normal saline, whereas Group III animals received gemfibrozil (10 mg/kg/day I.P, suspended in gum acacia in water) and Groups IV and V animals received extract by oral route in doses of 200 mg/kg/day and 400 mg/kg/day, respectively, throughout the 8 days experiment. After the experimental period, the overnight fasted experimental rats were sacrificed by decapitation under light ether anesthesia and blood was collected. Serum was separated, and lipid profiles (biochemical parameters) were analyzed. The lipid profiles of dexamethasone-induced hyperlipidemia model and the results of the antihyperlipidemic effect of extract treated groups of dexamethasone-treated groups were shown in Table 1.

Statistical evaluation

All the values were expressed as mean ± standard error of mean. The data were statistically analyzed by one-way ANOVA followed by Dennett's *t*-test, and value $P < 0.05$ was considered to be significant.

RESULTS

The present study was carried out to assess the antihyperlipidemic effect of EETS against dexamethasone-induced hyperlipidemia in male Wister rats. When EETS was evaluated for its antihyperlipidemic activity against dexamethasone-induced hyperlipidemia model, it showed a statistically significant activity at doses of 200 and 400 mg/kg by oral administration. After 8 days treatment of dexamethasone, a significant rise in lipid and lipoprotein levels were observed in serum in dexamethasone-induced group when compared to the normal group. The results were depicted in Table 1.

Effect of EETS in biochemical parameters in serum

Effect on total cholesterol and total TGs

Total cholesterol levels in the hyperlipidemia-induced group have significantly increased compared to normal rats. The values have risen to 116.83 ± 1.686 mg/dl compared to Group I (normal rat group), in which values lie in the range 64 ± 1.351 mg/dl. This indicates hypercholesterolemia. In the treatment group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values are reduced to 82 ± 2.306 ($P < 0.001$) and 78.0 ± 2.386 mg/dl ($P < 0.01$), respectively. There is a significant reduction in total cholesterol values in EETS treatment group. On the other hand, gemfibrozil also has significantly reduced serum total cholesterol levels to 70.50 ± 1.351 mg/dl ($P < 0.001$) [Table 1].

The TG levels have reached as 150.83 ± 1.666 mg/dl in dexamethasone-induced group compared to normal rats where the values are 62.83 ± 1.776 mg/dl. This indicates triglyceridemia. In the group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values are significantly reduced to 77.16 ± 1.686 mg/dl ($P < 0.01$) and 73.16 ± 1.686 mg/dl ($P < 0.01$), respectively. In the gemfibrozil treated group, the values are reduced to 66.33 ± 0.763 mg/dl ($P < 0.001$) [Table 1].

Effect on phospholipids

Phospholipids are amphipathic lipid constituents of a membrane. They play an essential role in the synthesis of plasma lipoproteins. They function in transduction of messages from cell-surface receptors to certain messengers that control cellular processes and as surfactants.[19] Phospholipid levels in the dexamethasone-induced group have significantly increased compared to normal rats. The values have risen to 131.1 ± 2.982 mg/dl compared to normal rat group, in which values lie in the range 91.73 ± 1.165 mg/dl. In the treatment group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values are reduced to 103.65 ± 1.776 mg/dl ($P < 0.01$) and 98.32 ± 1.720 mg/dl ($P < 0.01$), respectively. There is a significant reduction in phospholipid values in EETS treatment group. On the other hand, gemfibrozil also has significantly reduced serum phospholipid levels to 94.37 ± 1.514 mg/dl ($P < 0.001$) [Table 1].

Effect on free fatty acid

Free fatty acid levels in dexamethasone-induced group have significantly increased compared to normal rats. The values have risen to 34.1 ± 0.151 mg/dl compared to normal rat group, in values lie in the range 19.37 ± 0.395 mg/dl. In the treatment group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values are reduced to 26.73 ± 0.306 ($P < 0.001$) and 25.37 ± 0.257 mg/dl ($P < 0.001$), respectively. There is a significant reduction in free fatty acid values in EETS treatment group. On the other hand, gemfibrozil also has significantly reduced serum free fatty acid levels to 21.62 ± 0.222 mg/dl ($P < 0.001$) [Table 1].

Effect on high-density lipoprotein (HDL) cholesterol

HDL-cholesterol in dexamethasone-induced group has significantly decreased compared to normal rats. The values have reduced to 26.16 ± 0.307 mg/dl compared to normal rat group, 37.66 ± 1.686 mg/dl. In the group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values were 23.33 ± 0.2 ($P < 0.01$) and 27.33 ± 0.332 mg/dl ($P < 0.01$), respectively. In gemfibrozil treated group, the values were 33.33 ± 0.420 mg/dl ($P < 0.001$) [Table 1].

Table 1: Effect of EETS against dexamethasone-induced hyperlipidemia in rats

Group	Treatment/dose	Total cholesterol (mg/dl)	Total TG (mg/dl)	HDL-cholesterol (mg/dl)	LDL-cholesterol (mg/dl)
I	Normal-control	64±1.351	62.83±1.776	37.66±1.686	12.66±0.332
II	Dexamethasone (10 mg/kg) S.C	116.83±1.686	150.83±1.666	25.16±0.306	53.33±1.686
III	Dexamethasone (10 mg/kg) S.C+gemfibrozil (10 mg/kg)	70.50±1.351*	66.33±0.763*	33.33±0.420*	20.66±0.32*

	P.O				
IV	Dexamethasone+EETS (200mg/kg)	82.0±2.306*	77.16±1.686*	23.33±0.32*	30.5±0.222
V	Dexamethasone+EETS (400mg/kg)	78.0±2.386*	73.16±1.686*	27.33±0.332*	26.5±0.222

Effect on LDL-cholesterol and VLDL-cholesterol

LDL-cholesterol in dexamethasone-induced group has significantly increased to 53.33 ± 1.686 mg/dl compared to normal rat group, 12.66 ± 0.332 mg/dl. In the group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values were reduced to 30.5 ± 0.222 mg/dl ($P < 0.001$) and 26.5 ± 0.220 mg/dl ($P < 0.001$), respectively. There is a significant reduction in LDL-cholesterol values in EETS treatment group. Gemfibrozil has significantly reduced LDL-cholesterol level to 20.66 ± 0.32 mg/dl ($P < 0.001$) [Table 1].

VLDL-cholesterol in dexamethasone-induced group has significantly increased to 37.33 ± 1.541 mg/dl compared to normal rat group, 12.16 ± 0.306 mg/dl. In the group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values are reduced to 28.33 ± 0.332 ($P < 0.01$) and 24.33 ± 0.332 mg/dl ($P < 0.01$), respectively. There is a significant reduction in EETS treatment group. Gemfibrozil has significantly reduced VLDL-cholesterol level to 17.16 ± 0.306 mg/dl ($P < 0.001$) [Tables 1 and 2].

Table 2: Effect of EETS against dexamethasone-induced hyperlipidemia in rats

Group	Treatment/dose	VLDL-cholesterol (mg/dl)	Atherogenic index	Phospholipids (mg/dl)	Free Fatty acid (mg/dl)
I	Normal-control	12.16±0.306	1.63	91.73±1.164	19.37±0.394
II	Dexamethasone (10 mg/kg) S.C	37.33±1.541	4.48	131.1±2.981	34.1±0.150
III	Dexamethasone (10 mg/kg) S.C+gemfibrozil (10 mg/kg) P.O	17.16±0.306**	2.19	94.37±1.513*	21.62±0.221*
IV	Dexamethasone+EETS (200mg/kg)	28.33±0.332**	3.39	103.65±1.775**	26.73±0.305*
V	Dexamethasone+EETS (400mg/kg)	24.33±0.332**	2.76	98.32±1.719**	25.37±0.256*

Effect on atherogenic index

Atherogenic index Total serum cholesterol Total serum HDL-c = holesterol. Atherogenic index in dexamethasone-induced hyperlipidemia control is increased to 4.48 compared to normal rat group, 1.63. In the group treated with EETS (200 mg/kg) and EETS (400 mg/kg), the values are significantly reduced to 3.39 and 2.76, respectively. Gemfibrozil has significantly reduced the values to 2.19 [Table 2].

DISCUSSION

Treatment with EETS produced a significant decrement in the serum level of lipids in the dexamethasone-induced hyperlipidemia in male Wistar rats. Beta-sitosterol a phytosterol is found to be useful in the treatment of hyperlipidemia. Hypolipidemic effect of proteins, gums, saponins, and beta-sitosterol have been reported by several authors. The EETS contains carbohydrates, glycosides, alkaloids, tannins, saponins, phenolic compounds, phytosterols, and flavonoids. The steroidal phytoconstituents may mimic the cholesterol at the formation of lipoproteins and chylomicrons. Moreover, many of the proven antihyperlipidemic drugs were possessing the similar pharmacophore with the cholesterol. The high amount of phytosterols and alkaloids may be responsible for the hypolipidemic effect. It was found that EETS was more effective in higher dose as compared to lower dose as an antihyperlipidemic agent against dexamethasone-induced hyperlipidemia model. It also improves that HDL-cholesterol levels and lower atherogenic index. Further experiments are required to prove the

mechanism and advantage of EETS over other drugs.

CONCLUSION

These results suggested that EETS possesses a significant antihyperlipidemic activity in dexamethasone-induced hyperlipidemia in male Wistar rats. The isolation of the active phytochemical constituents is underway.

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