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Antihyperlipidemic activity of Plectranthus vettiveroides roots

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ABSTRACT

Plant based Natural ingredients are important in the prevention and treatment of a variety of diseases and disorders in humans. Hyperlipidemia is a key clinical cause in heart disease and diabetes. Plectranthus vettiveroides, a member of the Lamiaceae family, was discovered to have anti-inflammatory, antibacterial, and anti-cancer properties, as well as CNS depressant properties. The current research uses an in vivo animal model to look at its antihyperlipidemic function. Dexamethasone administration causes a substantial rise in serum cholesterol and triglyceride (TG) levels, as well as an increase in the atherogenic index, in rats. The treatment of dexamethasone-induced hyperlipidemia in rats with an ethanolic extract of P.vettiveroides root (200 and 400 mg/kg) showed substantial inhibition by keeping cholesterol, TGs, and other serum levels near average.

Keywords: *Plectranthus vettiveroides*, Cholesterol, Triglyceride, HDL, VLDL

INTRODUCTION

Herbal medicines are used by about 80% of the world's population for primary health care, mainly in developing countries. [1] They remained stationary due to their protection, effectiveness, cultural acceptability, better compatibility with the human body, and less side effects. In ancient literatures, herbal remedies were listed for age-related diseases including memory loss, diabetic wounds, osteoporosis, and immune and liver disorders, for which no modern medicine or only palliative therapy was available. Digoxin, morphine, emetine, aspirin, and ephedrine, among the life-saving and basic drugs discovered from medicinal plants, were known to modern therapeutics several centuries earlier. Namdeo made a point regarding secondary metabolites derived from plants, claiming that about a quarter of all suggested pharmaceuticals in developed countries contain compounds derived from plants, either directly or indirectly. [2] There is a widespread misconception that green medicine is both healthy and reliable.

Plant-based drugs have piqued people's interest in recent years. Many pharmaceutical companies are currently conducting comprehensive research on plant materials in order to determine their therapeutic value. [3] According to the World Health Organization, 4 billion people (or 80% of the global population) use plant-derived products for any form of primary health care. Approximately 74 percent of the 119 plant-derived medicines used today are directly linked to their common use as plant medicines by native cultures. Atherosclerosis is one of the leading causes of death worldwide in both developed and developing countries such as India. [4] One of the key risk factors for atherosclerosis is elevated levels of low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) associated with cholesterol and triglycerides (TGs). We can treat hyperlipidemia by targeting the atherogenic pathway, which is one of the palliative treatment options for atherosclerosis. (5) There are several allopathic antihypolipidemics on the market, but due to their side effects and contraindications, they are not widely used. Herbal hypolipidemics have recently gained popularity as a way to fill the voids. [6]

Plectranthus vettiveroides is also known as *Coleus vettiveroides, Coleus zeylanicus, Plectranthus zeynanicus* (Lamiaceae). The main phytochemical components of Iris are diterpenoids, essential oils and phenols. About 140 diterpenes were identified from the colored leaf glands of Platycladus species. The main components of Jerusalem artichoke essential oil are mono and sesquiterpenes. Flavonoids seem to be rare in *Platycladus orientalis*, only two flavonoids have been identified, 4',7-dimethoxy-5,6-cone in *Platycladus orientalis*, thus obtaining viologen from *P.marruboides* And golden chicken essence. Traditionally, it has been used as an antibacterial, deodorant, and cooling agent. It has also been used to prevent headaches and fever from burning eyes. The purpose of this research is to study the antihyperlipidemic activity of root of *Plectranthus vettiveroides*.

MATERIALS AND METHODS

Chemicals

SD Fine Chemicals India Ltd. provided the study's solvents (petroleum ether and ethanol), which were of laboratory grade. All other chemicals used in this study were purchased from Merck, India, and were of analytical grade. Gemfibrozil was purchased from Medindia, and dexamethasone and all other chemicals used in this study were purchased from Merck, India.

Plant materials

Plant roots of Plectranthus vettiveroides were collected in Namakkal, Tamilnadu, India, in June 2020. The plant was then botanically described and authenticated by a botanist at the Coimbatore Botanical Surveyor.

Experimental animals

The research used male Wistar rats (150-180 g) of about the same age obtained from Elite labs in Hyderabad, India. They were housed in polypropylene animal cages and given ad libitum water and a standard rodent pellet diet (Hindustan Lever Limited, Hyderabad). All of the animals were subjected to a 12-hour period of darkness followed by 12-hours of light. The animals were fasted for at least 12 hours before each test. All of the tests were done in the morning, in accordance with existing laboratory animal care guidelines and ethical guidelines for investigating experimental pain in conscious animals. In laboratory animals, a traditional oral gastric cannula and syringe were used to administer drugs.

Methodology

Extraction

The plant roots were dried in the shade and coarsely powdered using a mechanical grinder after the moisture content limits were verified. The powder was then sieved with a No. 40 sieve and placed in an airtight container for extraction.

Ethanol extract of P.vettiveroides [7]

P.vettiveroides' dried root was powdered and extracted with 95 percent ethanol at 75-78°C for 48 hours. The solvent was distilled out of the crude extract after the extraction was done. The residue was a dark brown colour. After that, the residue was concentrated and deposited in desiccators.

Pharmacological evaluation

Dexamethasone-induced hyperlipidemia model

Elevated glucocorticoid hormonal levels increase plasma lipid concentration, but this varies by species. The injection of glucocorticoids into rats stimulates the synthesis of triacylglycerol in the liver, which may contribute to the accumulation of fatty liver. The stimulation of TG output may lead to an increase in VLDL secretion. Dexamethasone injections for several days in rats have been shown to increase VLDL secretion. Hyperlipidemia is caused by an imbalance in lipid metabolism caused by a rise in TG levels. Dexamethasone therapy for four days in newborn rats resulted in a widespread rise in serum lipids. [8-11]

Dexamethasone-induced hyperlipidemia in rats

Induction of hyperlipidemia

Dexamethasone, a glucocorticoid believed to cause plasma lipid elevation, may be used to induce hyperlipidemia. To induce hyperlipidemia, rats were given dexamethasone (10 mg/kg/day, subcutaneous) for 8 days.

Procedure

To cause hyperlipidemia, all of the animals in Groups II, III, IV, and V were given a subcutaneous injection of dexamethasone (10 mg/kg/day, S.C) for 8 days. Throughout the 8-day experiment, the animals in the standard and hyperlipidemic control groups were given normal saline, while Group III animals were given gemfibrozil (10 mg/kg/day I.P, suspended in gum acacia in water) and Groups IV and V animals were given extract by oral route in doses of 200 mg/kg/day and 400 mg/kg/day, respectively. The overnight fasted experimental rats were sacrificed by decapitation under light ether anaesthesia after the experiment time, and blood was collected. Lipid profiles (biochemical parameters) were analysed after the serum was isolated. Table 1 shows the lipid profiles of the dexamethasone-induced hyperlipidemia model as well as the effects of the antihyperlipidemic effect of extract-treated dexamethasone-treated classes.

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 aim of this study was to see how effective EEPV is at reducing dexamethasone-induced hyperlipidemia in male Wistar rats. When tested for antihyperlipidemic activity in a dexamethasone-induced hyperlipidemia model, EEPV showed statistically significant activity at doses of 200 and 400 mg/kg given orally. When comparing the dexamethasone-induced group to the normal group, a substantial increase in serum lipid and lipoprotein levels was observed after 08 days of dexamethasone therapy. Table 1 summarises the findings.

Effect of EEPV in biochemical parameters in serum

Effect on total cholesterol and total TGs

When compared to normal rats, total cholesterol levels in the hyperlipidemia-induced community have increased significantly. In comparison to Group I (normal rat group), where values range from 64 to 1.351 mg/dl, the values have risen to 116.83 1.686 mg/dl. This is a symptom of hypercholesterolemia. The values are reduced to 82 2.306 (P 0.001) and 78.0 2.386 mg/dl (P 0.01) in the EEPV (200 mg/kg) and EEPV (400 mg/kg) treatment groups, respectively. Total cholesterol levels in the EEPV treatment community are significantly lower. Gemfibrozil, on the other hand, substantially decreased serum total cholesterol levels to 70.50 1.351 mg/dl (P 0.001) [Table 1].

In the dexamethasone-induced group, TG levels reached 150.83 1.666 mg/dl, while normal rats had values of 62.83 1.776 mg/dl. Triglyceridemia is shown by this. The values are slightly lower in the EEPV (200 mg/kg) and EEPV (400 mg/kg) groups, with 77.16 1.686 mg/dl (P 0.01) and 73.16 1.686 mg/dl (P 0.01), respectively. The values in the gemfibrozil-treated group are lower, at 66.33 0.763 mg/dl (P 0.001) [Table 1].

Effect on phospholipids

Membrane phospholipids are amphipathic lipid constituents. They are essential for the production of plasma lipoproteins. They serve as surfactants and transducers of messages from cell-surface receptors to specific messengers that regulate cellular processes. When compared to standard rats, the dexamethasone-induced community had significantly higher phospholipid levels. In comparison to the standard rat population, where values range from 91.73 to 1.165 mg/dl, the values have risen to 131.1 2.982 mg/dl. The values are reduced to 103.65 1.776 mg/dl (P 0.01) and 98.32 1.720 mg/dl (P 0.01) in the EEPV (200 mg/kg) and EEPV (400 mg/kg) treatment groups, respectively. The phospholipid levels in the EEPV treatment community are significantly lower. Gemfibrozil, on the other hand, substantially decreased serum phospholipid levels to 94.37 1.514 mg/dl (P 0.001). [Fig:1]

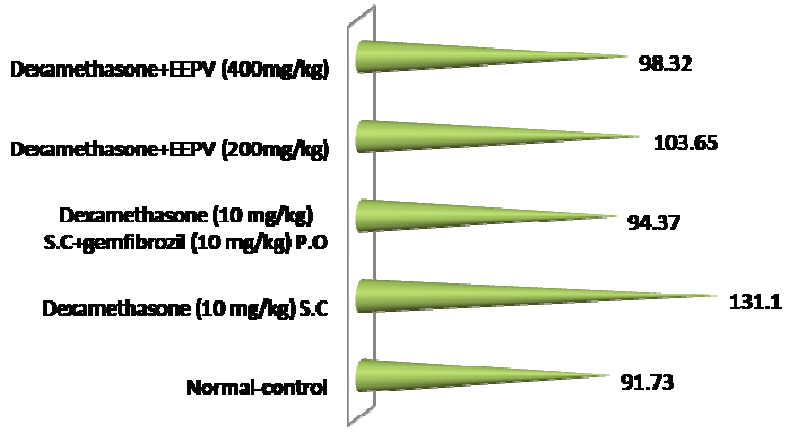

Figure 1: Effect on phospholipids

Effect on free fatty acid

When compared to standard rats, the free fatty acid levels in the dexamethasone-induced group were significantly higher. In comparison to the standard rat population, where values range from 19.37 to 0.395 mg/dl, the values have risen to 34.1 0.151 mg/dl. The values are reduced to 26.73 0.306 (P 0.001) and 25.37 0.257 mg/dl (P 0.001) in the EEPV (200 mg/kg) and EEPV (400 mg/kg) treatment groups, respectively. In the EEPV treatment group, free fatty acid values are significantly lower. Gemfibrozil, on the other hand, substantially decreased serum free fatty acid levels to 21.62 0.222 mg/dl (P 0.001). [Figure:2].

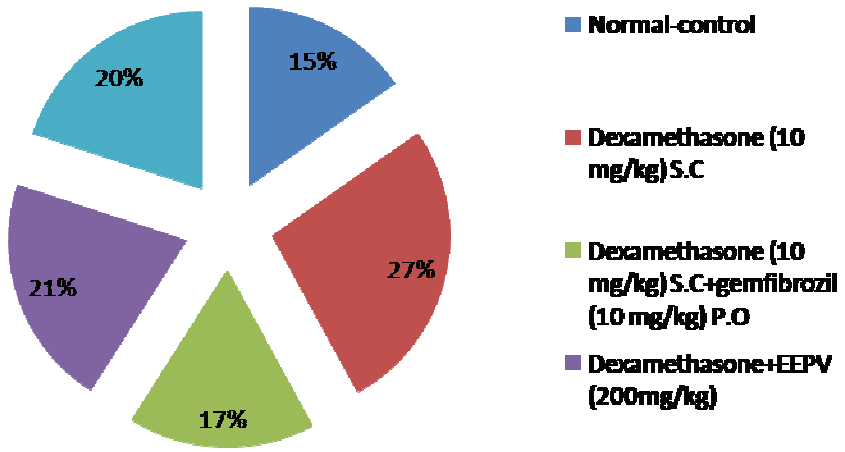

Figure 2: Effect on free fatty acid

Effect on high-density lipoprotein (HDL) cholesterol

When compared to normal rats, HDL cholesterol levels in the dexamethasone-induced community were significantly lower. As compared to the standard rat group's 37.66 1.686 mg/dl, the values have dropped to 26.16 0.307 mg/dl. The values were 23.33 0.2 (P 0.01) and 27.33 0.332 mg/dl (P 0.01) in the EEPV (200 mg/kg) and EEPV (400 mg/kg) classes, respectively. The values in the gemfibrozil-treated category were 33.33 0.420 mg/dl (P 0.001) [Table 1].

Table 1: Effect of EEPV 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) P.O	70.50±1.351*	66.33±0.763*	33.33±0.420*	20.66±0.32*
IV	Dexamethasone+EEPV (200mg/kg)	82.0±2.306*	77.16±1.686*	23.33±0.32*	30.5±0.222
V	Dexamethasone+EEPV (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 levels in the dexamethasone-induced group increased dramatically to 53.33 1.686 mg/dl, compared to 12.66 0.332 mg/dl in the normal rat group. The values were reduced to 30.5 0.222 mg/dl (P 0.001) and 26.5 0.220 mg/dl (P 0.001) in the EEPV (200 mg/kg) and EEPV (400 mg/kg) groups, respectively. In the EEPV treatment community, LDL cholesterol levels are significantly lower. [Table 1] shows that gemfibrozil significantly decreased LDL cholesterol levels to 20.66 0.32 mg/dl (P 0.001).

VLDL-cholesterol levels in the dexamethasone-induced group increased dramatically to 37.33 1.541 mg/dl, compared to 12.16 0.306 mg/dl in the control group. The values are reduced to 28.33 0.332 (P 0.01) and 24.33 0.332 mg/dl (P 0.01) in the EEPV (200 mg/kg) and EEPV (400 mg/kg) classes, respectively. In the EEPV treatment community, there is a substantial decrease. Gemfibrozil decreased VLDL cholesterol levels to 17.16 0.306 mg/dl (P 0.001), a substantial reduction. [Figure:3]

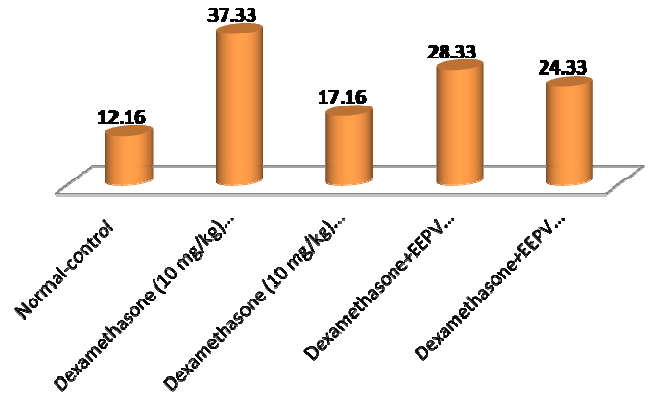

Figure 3: Effect on LDL-cholesterol and VLDL-cholesterol

Effect on atherogenic index

The atherosclerotic index is a measure of how Total cholesterol in the blood HDL-c = holesterol in total serum. As compared to the normal rat population, the atherogenic index in dexamethasone-induced hyperlipidemia regulation is increased to 4.48. The values in the EEPV (200 mg/kg) and EEPV (400 mg/kg) groups are slightly lower, at 3.39 and 2.76 mg/kg, respectively. [Figure:4] Gemfibrozil greatly decreased the values to 2.19.

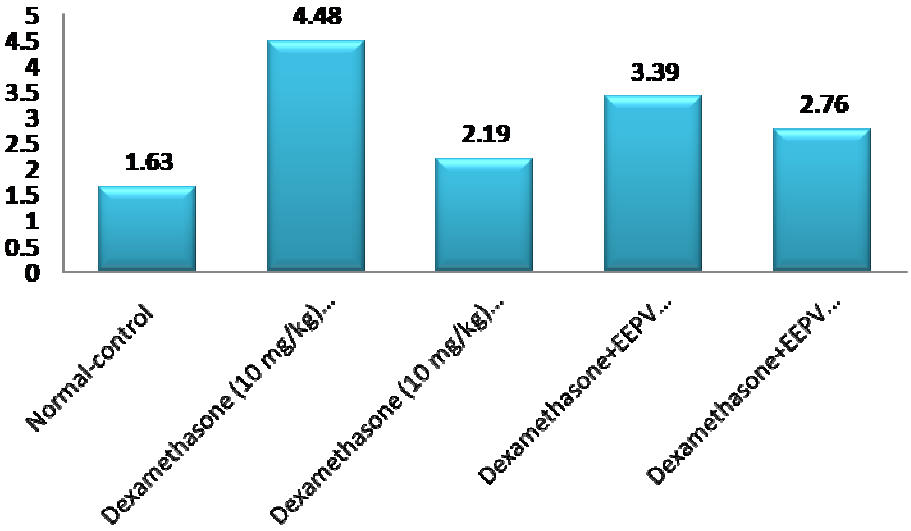

Figure 4: Effect on atherogenic index

DISCUSSION

In male Wistar rats with dexamethasone-induced hyperlipidemia, treatment with EEPV resulted in a substantial reduction in serum lipid levels. The phytosterol beta-sitosterol has been discovered to be beneficial in the treatment of hyperlipidemia. Several authors have stated that proteins, gums, saponins, and beta-sitosterol have a hypolipidemic effect. Carbohydrates, glycosides, alkaloids, tannins, saponins, phenolic compounds, phytosterols, and flavonoids are all included in the EEPV. At the formation of lipoproteins and chylomicrons, steroidal phytoconstituents can act like cholesterol. Furthermore, many of the proven antihyperlipidemic drugs had a pharmacophore that was identical to cholesterol. The hypolipidemic effect may be due to the high volume of phytosterols and alkaloids. In a dexamethasone-induced hyperlipidemia model, higher doses of EEPV were found to be more effective than lower doses as an antihyperlipidemic agent. It also raises HDL cholesterol levels and reduces the atherogenic index. More research is needed to prove the mechanism of EEPV and its advantages over other drugs.

CONCLUSION

These findings indicated that EEPV has antihyperlipidemic activity in male wistar rats with dexamethasone-induced hyperlipidemia. The active phytochemical constituents are currently being isolated.

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