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Hypocholesterolemic effect of *Enicostemma axillare* L., on fructose induced Heart disease in swiss albino rats

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ABSTRACT

The antioxidant effect of the ethanolic extract of *Enicostemma axillare*, an indigenous ayurvedic medicinal plant used in India, was studied in rats with fructose-induced cardiac diseases. Rats were divided into four groups: Groups 1 received Normal feed with water. Groups 2 received Fructose treated (The oral administration of 10g of fructose/ 100ml of distilledv water/kg body weight for 21 days), Groups 3 received Fructose and low dose of *Enicostemma axillare* (150mg/ kg B.wt.).Groups 4 received Fructose and high dose of *Enicostemma axillare* treated [oral administration of fructose (10g/ 100ml distilled /kg b. wt) and *Enicostemma axillare* (250mg/ kg B.wt)]. For 21 days. The results showed significantly elevated level of serum and tissue thiobarbituric acid reactive substances, and significantly lowered activities/levels of antioxidants such as, catalase, and glutathione-S-transferase and reduced glutathione in fructose -treated rats compared with control rats. Administration of ethanolic extract of *Enicostemma axillare* to rats with fructose -induced liver injury significantly decreased the levels of serum and tissue thiobarbituric acid reactive substances and significantly elevated the activities of catalase, glutathione-S-transferase and reduced glutathione in the tissues compared with unsupplemented fructose -treated rats. These findings suggest that ethanolic extract of *Enicostemma axillare* has a modulatory effect on fructose -induced hepatotoxicity in rats.

Keywords: Antioxidant, *Enicostemma Axillare*, Lipid Profile and Fructose

INTRODUCTION

The Heart is pear shaped muscular organ vertebrates responsible for pumping blood through the blood vessels by repeated rhythmic contractions. The term cardiac (as in cardiology) means “related to the heart” and comes from the Greek, () kardia for “Heart”. The heart is composed

of cardiac muscle, an involuntary muscle tissue, which is found only within this organ ^[1]. Heart disease is the leading cause of death, many of the risk factors for heart disease are influenced are of life style. For example, smoking, lack of exercise and consumption of a high fat diet all contribute to risk. A healthy diet is important for both prevention and treatment of cardiovascular disease ^[2].

Hypercholesterolemia (Literally: high blood cholesterol) is the presence of high levels of cholesterol in the blood. It is not a disease, but a metabolic derangement that can be secondary to much disease and can contribute to many form of disease, mostly notably cardiovascular disease [3]. Atherosclerosis can be defined as the “Focal pathological phenomenon characterized by thickening, hardening, hardening of arteries due to accumulation of lipid, carbohydrates, blood products, fibrous tissue and calcium deposits within the subendothelial space [4].

The characteristic feature of atherosclerosis is the buildup of fatty deposits called Deposits called “plaque”, along the inner artery walls. Subsequently leading to narrowing of the arterial passage way and eventually impaired circulation. [5] The atherosclerosis can cause blood clots to form that will ultimately stop blood flow. If this happens in the arteries supplying the heart is a stroke where a portion of brain tissue dies.

Hypercholesterolemia is silent. There are no symptoms that are obvious to the naked eye. It is diagnosed by a blood test or after a heart attack or stroke occurs. High plasma cholesterol also impairs the functional integrity of coronary vasculature and increases ischemia- reperfusion injury to the Heart [6]. Increased intake of a food may be related to oxidative stress [7] an imbalance between oxidant and antioxidant system in favor of the formes [8]. Recent research in our laboratory has shown that increased caloric intake is an important factor decreasing the mitochondrial membrane fluidity and increasing the reactive oxygen species generation [9].

Fructose is readily absorbed from the diet and rapidly metabolized principally in the liver. Fructose can provide carbon atoms for the both the glycerol and the acyl portions of triglyceride. Fructose is thus a highly efficient inducer of denovo lipogenesis. High concentrations of fructose can serve as a relatively unregulated source of acetyl CoA. In contrast to glucose, dietary fructose does not stimulate insulin or leptin (which are both important regulators of energy intake and body adiposity). Stimulated triglyceride synthesis is likely to lead to hepatic insulin sensitivity, as well as the increased formation of VLDL particles due to higher substrate availability, increased apoB stability, and MTP, the critical factor in VLDL assembly.

There are numerous studies in which dietary fructose has been shown to induce hyperlipidemia in rodents [10] Herman et al. [11] reported that’s fed a high- fructose diet had sustained elevation in serum triacylglycerol. Increases in VLDL secretion can then lead to chain in other lipoprotein and lipids, such as LDL, TG, and Cholesterol. In this context’ Kelley et al., [12] hypothesized that pro –oxidant stress response pathways may mediate hepatic increases in VLDL secretion and delayed clearance upon fructose feeding. The presence investigation has been undertaken to find out the pharmacological effects of the Hydroalcoholic extract of *Enicostemma axillare* which studies evaluated the potential of hypocholesterolemic and antioxidant activity.

Swertiamarin, gentiocrucine, enicoflavine, genkwanin, isovitexin, swortistin, sapanarin, 5-oglycosyliswertisin, gentiocrycine, swertiamarin tetra acetate, 3- acyl- 3,4 dehydrogentiopicroside, ophelic acid, n-hexacosnal, hepatocosane, non acosane, myristic acid, stearic acid, oleic acid, gentianine , betulin, alkaloids. The plant, *E. axillare* is a bitter tonic, acrid, thermogenic, digestive, carminative, stomachic, laxative, anthelmintic, anti-inflammatory, liver tonic, Astringent, colic, belminthiasis, abdominal ulcers, hernia constipation, dropsy, swelling, liver disorders, glycosuria, leprosy, skin disease, pruritus, intermittent fever and malaise.

This study was designed to test the hypothesis that *Enicostemma axillare* would reduce fructose induced toxicity by lipid profile.

MATERIALS AND METHODS

Plant material

The leaves of *Enicostemma axillare* were collected from the S.T.E.T Medical plant garden, Mannargudi, Thiruvavur District and authenticated by the Botany Department of A.V.V.M. Sri Pushpam College, Poondi. After authentication the plant material was washed under running tap water.

Preparation of Plant Extract

Enicostemma axillare leaves were dried (without direct sunlight) and converted into powder form. The powder obtained was successively extracted in methanol and distilled water by using soxhlet apparatus. It was stored at 4°C until used

when needed the residual extract was suspended in distilled water and used in the study.

Animals

A healthy swiss albino rats were housed in well ventilated hygienic atmosphere. Animals with 200 – 300g were using our study. Animals were fed with commercial rat feed (Saidurga feeds & foods, Bangalore) and tap water adlibitum. After randomization into various groups, the rats were acclimatized for a period of 2-3 days in the new environment before initiation of experiment.

Chemicals

All of the chemicals were of analytical grades and were obtained from Central Drug House Pvt. Ltd (New Delhi, India).

Experiment design

In the experiment, a total of 16 rats was used. The rats were divided into following 4 groups of 6 each.

Group I : Normal feed with water

Group II : Fructose treated (The oral administration of 10g of fructose/ 100ml of distilled water/kg body weight for 21 days),

Group III : Fructose and low dose of *Enicostemma axillare* (150mg/ kg B.wt.).

Group IV : Received Fructose and high dose of *Enicostemma axillare* treated [oral administration of fructose (10g/ 100ml distilled /kg b. wt) and *Enicostemma axillare* (250mg/ kg B.wt.).

Sample Collection

After 21 days of herbal treatment, the blood sample was collected from the anaesthetized rats by puncturing the orbital sinus. After the collection of blood, it was allowed to stand for 10 mts.

Biochemical measurements

Lipoproteins were separated by the method of Nerurkar and Tarkar^[13], Freidwald formula^[13].

Tissue and plasma Total cholesterol^[14], and Triglyceride^[15] were determined.

Statistical analysis

Results are expressed as mean \pm SE from six observations

RESULT

Our research is focus on the antioxidant activity of *Enicostemma axillare* which is evaluated by assessing the Biochemical parameters such as and Antioxidant enzymes namely, cholesterol, TG, LDL, HDL, were determined in the plasma and Heart. The results clearly pin points the hypocholesterolemic of *E. axillare* in hypercholesterolemic rats.

Table I represents the level of lipid profile in our experimental study. The cholesterol, TG, LDL and VLDL level of lipid profile in plasma was markedly increased in the induced group rats, when compared to normal group. Then the level was decreased in the hydroalcoholic extract of high dose (250mg/kg) treated group and low dose (150mg/kg) treated group. The level of HDL showed reverse effect of above parameters. The level is increased after the administration of low dose treated groups. The high dose (250 mg/kg) treated groups showed significant results when compared to low dose treated groups.

Table II represents the level of lipid profile in our experimental study. The cholesterol, TG, LDL and VLDL level of lipid profile heart was markedly increased in the induced group rats, when compared to normal group. Then the level was decreased in the hydroalcoholic extract of high dose (250mg/kg) treated group and low dose (150mg/kg) treated group. The level of HDL showed reverse effect of above parameters. The level is increased after the administration of low dose treated groups. The high dose (250 mg/kg) treated groups showed significant results when compared to low dose treated groups.

TABLE 1 Table showing level of lipid profile in plasma

S.No	Groups	Cholesterol (mg/dl)	TG (mg/dl)	HDL (mg/dl)	VLDL(mg/dl)	LDL(mg/dl)
1.	GP-I	65.3 \pm 1.2	70.8 \pm 1.3	21.8 \pm 0.2	14.16 \pm 0.1	29.3 \pm 0.2
2.	GP-II	175.2 \pm 10.5	175.3 \pm 9.5	13.3 \pm 0.1	35.06 \pm 0.3	126.8 \pm 0.1
3.	GP-III	125.3 \pm 8.3	85.4 \pm 6.5	18.3 \pm 0.2	17.08 \pm 0.1	89.92 \pm 0.3
4.	GP-IV	80.2 \pm 5.5	70.2 \pm 5.2	19.2 \pm 0.3	14.04 \pm 0.1	52.96 \pm 0.2

(Values are mean \pm S.E from 6 rats in each group).

TABLE 2 Table showing level of lipid profile in heart

S.No	Groups	Cholesterol (mg/dl)	TG (mg/dl)	HDL (mg/dl)	VLDL(mg/dl)	LDL(mg/dl)
1.	GP-I	4.2± 0.2	8.8± 0.3	2.9± 0.2	1.76±0.1	0.46±0.03
2.	GP-II	12.1± 0.5	19.3± 1.5	2.1± 0.1	3.86± 0.3	6.14± 0.5
3.	GP-III	7.3± 0.3	12.4±1.0	1.9±0.2	2.48± 0.2	2.92±0.3
4.	GP-IV	6.2±.5	17.3±0.2	2.8±0.3	3.46±0.2	0.06±0.02

(Values are mean ± S.E from 6 rats in each group).

DISCUSSION

A number of indigenous formulations been derived to possess hypocholesterolemic activity. A few of them have also been studied in experimental models. The present study was designated primarily to evaluate the hypocholesterolemic activity *Enicostemma axillare* in hypercholesterolemia rats induced by fructose.

Elevated level of circulatory cholesterol because deposit inside blood vessel these deposit called plaque, are composed of fats from the blood stream. When the deposits become sufficiently large, they block blood vessels and decrease the blood flow. These deposits result in a disease process called atherosclerosis. High plasma cholesterol also impairs the functional integrity of the coronary vasculature and increases ischemia – reperfusion injury to the heart. [16]

An increasing body of evidence indicates that the metabolism of TG is a strong predictor of CAD and is linked to atherosclerosis, although the relation appears. To be both statistically and biologically more complex than the relation between plasma cholesterol and coronary artery disease risk. TG excess of fat diet increased the TG level which is one of the causes for Hardening of arteries. Although there are many links between high TG levels and coronary heart disease, Our understanding of the risk was poor, so that reducing levels has not been advocated and is much less important than reducing LDL- Cholesterol levels.

VLDL production is directly related to the body fat. [17] Severe elevation in the VLDL cholesterol leads to hypercholesterolemia [18]. VLDL is the main carrier of triglycerides and it is less harmful than LDL, but still can damage the arterial lining. LDL is harmful and predisposes to premature atherosclerosis. LDL cholesterol is the primary focus for diagnosis and treatment guidelines of the US national cholesterol Education program

(NECP). Elevated LDL and cholesterol are the major risk factors for the development of coronary artery disease [19]. Oxidation of LDL is injured in both endothelium and smooth muscle *in vitro* and has been found in both human and in experimental lesions of the atherosclerosis. An oxidative modification of LDL appears to have an important role in coronary artery disease and atherosclerosis. Reduction in LDL and increase in HDL are significantly related to lipid lowering therapy [20]. Elevated level of HDL is associated with a decreased atherosclerosis tendency. HDL particles are considered antiatherogenic. A strong inverse relation between HDL-C concentration and risk factor for coronary artery disease. The complete concentration and risk factor for Coronary artery disease. The complete absence of HDL leads to serve premature atherosclerosis.

CONCLUSION

Thus, our study shows that *Enicostemma axillare* effectively protects the tissues against fructose-induced hyperlipidemia. They offer a safer alternative to synthetic chemicals and can be obtained at a very low cost. *Enicostemma axillare* can be used for effective protection of heart disorders, their potential under field conditions needs to be evaluated. Further investigation regarding the heart protective principles of *Enicostemma axillare* should be carried out in future.

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