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EVALUATION OF ANTIHYPERLIPIDEMIC ACTIVITY OF *SAPINDUS EMARGINATUS* IN RATS

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Abstract:

Obesity and hyperlipidemia have become major disorders predominantly causing prevailing cardiovascular diseases and ultimately death. The prolonged use of anti-obesity drugs and statins for reducing obesity and blood lipid levels is leading toward adverse effects of kidneys and muscles, specifically rhabdomyolysis. The objective of this study is to evaluate potential of seeds of Sapindus emarginatus against hyperlipidemia. In this model of Hyperlipidemia, 30 adult male wistar rats (200-250gms) were evenly divided into 5 groups in both groups. Group-1 and Group-2 served as untreated and model controls respectively, while Group-3, 4 and 5 were the treatments groups which were simultaneously treated with standard, 100 and 200 mg/kg extract respectively along with High Fat Diet. On last day, blood samples for biochemical parameters, were obtained under inhaled diether anaesthesia. The outcomes of this study were expressed as mean standard error and data were evaluated by using analysis of variance followed by multiple comparisons. Oral administration of 100 mg/ kg and 200mg/kg body weight of Methanolic extract residual fraction of Moringa oleifera. Leaves exhibited a significant reduction (P < 0.01) in serum lipid parameters such as triglycerides, total cholesterol, low density lipoprotein (LDL), very LDL and increase in high density lipoprotein in hyperlipidemic rats when compared with hyperlipidemic control in both models. Our results demonstrated that Methanolic extract fraction of Sesbania grandiflora. Possessed significant antihyperlipidemic activity.

Keywords: *Sesbania grandiflora, Cholesterol, LDL, triglycerides and antihyperlipidemic activity.*

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INTRODUCTION

Hyperlipidemia is a condition when abnormally high levels of lipids i.e. the fatty substance are found in the blood. This condition is also called hypercholesterolemia/hyperlipoproteinemia[1]. Human body is complex machinery and for maintaining the homeostasis of various organ and organ system. Any undesirable change will disturb the balance resulting in diseased state[2]. Lipids are fats in the blood stream, commonly divided into cholesterol and triglycerides. Cholesterol circulates in the bloodstream and is involved in the structure and function of cells. Triglycerides(TG) are best viewed as energy that is either used immediately or stored in fat cells.TG are manufactured in the liver from the foods or by being absorbed from the intestine[3]. Virchow in 19thcentury who identified cholesterol crystals in atherosclerotic lesion and stated that endothelial cell injury initiates atherogenesis. In a modification of this hypothesis it was proposed that the endothelium normally influences the behaviour of arterial smooth muscle cells by providing a barrier to the passage of plasma proteins,and that the major effect of haemodynamic or other factors that injure endothelium is to reduce the effectiveness of the barrier[4].Arteries are normally smooth and unobstructed on the inside, but in case of increased lipid level, a sticky substance called plaque is formed inside the walls of arteries. This leads to reduced blood flow, leading to stiffening and narrowing of the arteries. It has been proved that elevated plasma levels of cholesterol and of LDL are responsible for atherosclerosis in man, and epidemiological data suggests that elevated plasma levels of HDL have a protective effect[5].

Classification of Lipid Concentrations

The cholesterol along with some other types of fats cannot be dissolved in the blood. Moreover, in order to be transported to and from cells, they have to be specially carried by certain molecules called lipoproteins, which consist of an outer layer of protein with an inner core of cholesterol and triglycerides [6,7]. In addition, the lipoproteins have been found essential for cholesterol to move around the body. The lipids can be classified as TC, triglycerides, LDL, HDL and very low density lipoprotein (VLDL) cholesterol.

Total cholesterol

According to guidelines of National Cholesterol Education Program (NCEP), TC concentrations below 200 mg/dL have been regarded as desirable, whereas, concentrations greater than 240 mg/dL are referred to as hyperlipidemic. However, epidemiological evidence suggests that the risk of cardiac events decreases as TC levels fall approximately to 150 mg/dL. Moreover, TC should be less than 180 mg/dL for children [8,9].

Triglyceride

Triglycerides are another type of fat that is carried in the blood by VLDL. The excess calories, alcohol or sugar in the body get converted into triglycerides and stored in fat cells throughout the body.The triglyceride concentration less than 150 mg/dL is regarded as normal, wheras, concentrations of 200-499 mg/dL are considered as high. Moreover, concentrations of 500 mg/dL or higher are considered dangerous for the development and progression of various CVDs [10].

LDL cholesterol

LDL is commonly known as the bad cholesterol, which is produced by the liver and carry cholesterol and other lipids from the liver to different areas of the body like muscles, tissues, organs and heart. The high levels of LDL indicate much more cholesterol in the blood stream than necessary and hence, increase the risk of heart disease [11].According to NCEP guidelines, LDL cholesterol concentrations below 100mg/dL are considered optimal, whereas concentrations in the range of 160-189 mg/dL are considered to the higher side.However, increasing evidence supports that normal human LDL cholesterol concentration can be as low as 50 to 70 mg/dL.Moreover, it has been comprehensively seen that the risk of CVDs decreases as LDL cholesterol concentration decreases.

HDL cholesterol

HDL is commonly referred to as the good cholesterol, which is produced by the liver to carry cholesterol and other lipids from tissues back to the liver for degradation [12].High levels of HDL cholesterol have been considered as a good indicator of a healthy heart. The concentrations of 60 mg/dL or higher have been considered as optimal, whereas, HDL concentrations below 40 mg/dL are considered as major risk factor for CVDs. However, HDL is often interpreted in the context of TC and LDL concentrations, and hence may be regarded as less significant when LDL is low.

VLDL Cholesterol

VLDL is similar to LDL cholesterol in the sense that it contains mostly fat and not much protein. VLDL cholesterol is the lipoproteins that carry cholesterol from the liver to organs and tissues in the body. They

are formed by a combination of cholesterol and triglycerides. Moreover, VLDLs are heavier than LDL, and are also associated with atherosclerosis and heart disease[13]

Hyperlipidemia can be broadly divided into:
Primary; hyperlipidemia is due to Single gene defect: It is familial and called as monogenic or genetic.
Polygenic gene defect Multiple genetic defect, dietary and physical activity are caused due to it.

TYPES OF HYPERLIPIDEMIA

Table no : 1

TYPE	DISORDER	CAUSE	OCCURANCE	ELEVATED PLASMA LIPOPROTEIN
I.	Familial lipoprotein lipase deficiency	Genetic	Very rare	Chylomicrons
IIa	Familial hypercholesterolemia	Genetic	Less common	LDL
IIb	Polygenic hypercholesterolemia	Multifactorial	Commonest	LDL
III	Familial dysbetalipoproteinemia	Genetic	Rare	IDL,Chylomicrons Remnants
IV	Hypertriglyceridemia	Multifactorial Genetic	Common	VLDL
V	Familial combined hyperlipidemia	Genetic	Less common	VLDL,LDL

LDL-low density lipoprotein,VLDL-very low density lipoprotein,IDL-intermediate density lipoprotein[14]

SECONDARY HYPERLIPIDEMIA
It is associated with diabetes, myxoedema, nephritic syndrome, chronic alcoholism, with use of drugs like corticosteroids, oral contraceptives, Beta blockers[15]

Hypercholesterolemia
Reasons
- Hypothyroidism
- Anorexia nervosa
- Acute intermittent porphyria
- Obstructive liver disease
- Nephrotic syndrome
- Drugs: Progestins, thiazide diuretics, glucocorticoids, betablockers, Isotretinion, protease inhibitors, cyclosporine, mirtazapine, sirolimus

Hypertriglyceridemia
Reasons
- Obesity
- Pregnancy
- Lipodystrophy
- Acute hepatitis
- Diabetis mellitus
- Ileal bypass surgery
- Glycogen storage disease
- Systemic lupus erythematosus
- Monoclonal gammopathy: multiple myeloma

Hypocholesterolemia
Reasons
- Malanutrition
- Malabsorption
- Chronic liver disease

Low HDL
Reasons
- Malanutrition
- Obesity

Causes Of Hyerlipidemia

The main cause of hyperlipidemia includes changes in lifestyle habits in which risk factor is mainly poor diet i.e with a fat intake greater than 40 percent of total calories, saturated fat intake greater than 10 percent of total calories; and cholesterol intake greater than 300 milligrams per day or treatable medical conditions[16] .The abnormal cholesterol levels are the result of an unhealthy lifestyle including taking high-fat diet and other lifestyle factors like being overweight, smoking heavy alcohol use and lack of exercise. Other factors include diabetes, kidney disease, pregnancy, and an underactive thyroid gland [17] . Other illnesses that may elevate cholesterol levels include polycystic ovary syndrome and kidney disease. The higher levels of female hormones like estrogen, have been noted to increase or change cholesterol levels. In addition, drugs like diuretics, beta-blockers and medicines used to

treat depression have also been reported to raise cholesterol levels. Another modifying factors in the development and progression of hyperlipidemia are age and gender. It has been shown that cholesterol levels rise as the person gets older [18]. Heredity has also been a modifying factor for the progression of hyperlipidemia as it has been noted that the genes partly determine the amount of cholesterol body makes [19]. Other factors that cause hyperlipdemia without any prevalence information includes the following.

Berardinelli-Seip congenital lipodystrophy – hyperlipidemia

A rare genetic disorder having heptomegaly, loss genetic disorder characterized by diabetes mellitus, loss of body fat, hepatomegaly, enlarged genitals, increased skeletal growth and other abnormalities[20]

Berardinelli-Seip congenital lipodystrophy, type 1 – hyperlipidemia

A rare genetic disorder caused by a defect on the AGPAT2 gene on chromosome 9q34.326 characterized by early-onset diabetes mellitus, loss of body fat, serious insulin resistance, high blood triglycerides and fatty liver

Berardinelli-Seip congenital lipodystrophy, type 2 – hyperlipidemia

A rare genetic disorder caused by a defect on the BSCL2 gene on chromosome 11q13 by early-onset diabetes mellitus, loss of body fat, serious insulin resistance, high blood triglycerides and fatty liver[21].

Cholestasis

It is a condition the bile flow from the liver to the duodenum is blocked. It is of two types first one is caused by mechanical blockage in the duct system which occur from a gallstone or malignancy and other type is metabolic cholestasis, in which disturbances in bile formation occur because of genetic defects or acquired as a side effect of many medications [22]

Chromosome 15q, deletion:

A rare chromosomal disorder which occurs because of deletion of genetic material from the long arm of chromosome 1529.

Neuropathy, hereditary motor and sensory, Okinawa type:

A dominantly inherited, slow-progressing motor and sensory nerve disease which primarily involves the proximal muscles (i.e. the muscles closest to the trunk of the body)[23].

Pathophysiology Of Hyperlipidemia

- The pathophysiology of hyperlipidemia can be studied under two headins, i.e., primary hyperlipidemia and secondary hyperlipidemia. The pathophysiology of primary hyperlipidemia involve that the idiopathic hyperchylomicronemia defect in lipid metabolism leads to hypertriglyceridemia and hyperchylomicronemia which is caused by a defect in lipoprotein lipase activity or the absence of the surface apoprotein CII.Moreover, hyperchylomicronemia in cats with autosomal recessive defect in lipoprotein lipase (LPL) activity showed the occurrence of primary hyperlipidemia[24].

- In secondary hyperlipidemia, the postprandial absorption of chylomicrons from the gastrointestinal tract occurs 30-60 min after ingestion of a meal containing fat that may increase serum triglycerides for 3-10 hours. The diabetes mellitus patients have been noted to possess low LPL activity which further caused high synthesis of VLDL cholesterol by the liver ultimately leading to hyperlipidemia. Moreover, hypothyroidism-induced low LPL activity and lipolytic activity has been noted to reduce hepatic degradation of cholesterol to bile acids. Furthermore, hyperadrenocorticism increased the synthesis of VLDL by the liver causing both hypercholesterolemia and hypertriglyceridemia. Liverdisease hypercholesterolemia has been noted to be caused by reduced excretion of cholesterol in the bile. Furthermore, in nephrotic syndrome, the common synthetic pathway for albumin and cholesterol causes low oncotic pressure ultimately leading to enhanced cholesterol synthesis [25].

- The response-to-injury hypothesis states that risk factors such as oxidized LDL, mechanical injury to the endothelium, excessive homocysteine, immunologic attack, or infection-induced changes in endothelial and intimal function lead to endothelial dysfunction and a series of cellular interactions that culminate in atherosclerosis. The eventual clinical outcomes may include angina, myocardial infarction, arrhythmias, stroke, peripheral

arterial disease, abdominal aortic aneurysm, and sudden death.

- Atherosclerotic lesions are thought to arise from transport and retention of plasma LDL through the endothelial cell layer into the extracellular matrix of the subendothelial space. Once in the artery wall, LDL is chemically modified through oxidation and nonenzymatic glycation. Mildly oxidized LDL then recruits monocytes into the artery wall. These monocytes then become transformed into macrophages that accelerate LDL oxidation.
- Oxidized LDL provokes an inflammatory response mediated by a number of chemoattractants and cytokines (e.g., monocyte colony-stimulating factor, intercellular adhesion molecule, platelet-derived growth factor, transforming growth factors, interleukin-1, interleukin-6).

MATERIALS AND METHODS

I. PLAT MATERIAL:

The leaves of plant *Sapindus emarginatus* were collected from local market Hyderabad.

EXPERIMENTAL ANIMALS:

Male Wistar rats weighing (180-220g) were provided by animal house of Sigma Institute of Clinical Research and Administration (SICRA Labs), Kukatpally, Hyderabad, India. They were housed in ventilated rooms at a temperature of 24±2°c with a 12h light/dark cycle and 54±5% relative humidity, maintained on standard pellet and water ad libitum throughout the experimental period. The animals were acclimatized for a period of one week. The experiments were carried out according to the guidelines of the committee for the purpose of control and supervision of experiments on animals (CPCSEA), New Delhi, India and approved by the Institutional Animal Ethical Committee (IAEC) of Sigma Institute of Clinical Research and Administration pvt.ltd. Hyderabad.

II. DRUGS AND CHEMICALS:

Sesbania grandiflora, all other chemicals and diagnostic kits were provided by Sigma Institute of Clinical Research and Administration.

III. PREPARATION OF EXTRACT:

The collected plant dried leaves powder using mixer grinder. The powdered plant material leaf (250gm) was extracted with methanol, by Maceration process. Finally, extracts were air dried at room temperature. 10.2% and 7.2% w/w extract thus obtained was subjected for evaluation of Hyperlipidemic activity in rats. The test samples of extracts were made in appropriate concentrations using distilled water prior to its use for animal studies.

IV. PHYTOCHEMICAL SCREENING:

Preliminary phytochemical investigation was carried out on Methanol extract of *Sapindus emarginatus leaves* for detection of various phytochemical by standard methods.

V. DETERMINATION OF ACUTE ORAL TOXICITY:

Healthy albino rats of either sex of 2-2½-months-old of body weighs 125-150 g were housed in polypropylene cages at 25±2°C with light dark cycle of 12 h in the Animal House of the study center are to be used for the study. It should be acclimatized for seven days. All animals are to be given with standard rat feed and water ad libitum. The experiments were performed after approval of the protocol by the minute of Institutional Animal Ethics Committee (IAEC) 887/Po/Re/S/2005 CPCSEA and animal care was taken as per the guidelines of Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India.

Development of cholesterol diet model

High cholesterol diet consisting of Cholesterol (1%), sodium cholate (0.5%), sucrose (30%), casein(10%), butter (5%) and standard chow diet (53.5%) ad libitum, respectively, for the initial period of 2 weeks. The composition and preparation of HCD as were described elsewhere.

Experimental design

High Cholesterol (HC)-Diet

Wistar rats weighing 200- 250 gm, were divided into5 groups of 6 animals each. The animals of all the groups except normal group were given a lipid diet consisting of 2% cholesterol, 1% cholic acid and 2 ml coconut oil with standard pellet diet for 30 days. The first group (Normal control) received normal saline orally for 30days. The second group (High cholesterol diet (HCD)- positive control) was given High cholesterol diet (HCD) while the third Group received Atorvastatin 10 mg/kg, fourth and fifth groups were treated with hydro alcoholic extract of *Sapindus emarginatus* (2100 mg/kg and 200mg/kg, p.o.) respectively, once a day for 30 days. The fifth group was treated with Atorvastatin suspension prepared with 0.5% CMC (10mg/kg; p.o.), once a day

for 30 days. After 30 days blood was collected by tail vein methods. The collected samples were centrifuged for 10 minutes at 2000 r.p.m. and serum samples so collected were used for various biochemical tests.

Group 1: Normal control.1% CMC

Group 2: Hyperlipidemic control (Vehicle 1 ml/100g/day p.o).

Group 3: Hyperlipidemic treated with Atorvastatin (10 mg/kg, b.w./day p.o).

Group 4: Hyperlipidemic treated with MESE (100 mg/kg, b.w./day p.o).

Group 5: Hyperlipidemic treated with MESE (200 mg/kg, b.w./day p.o).

PARAMETERS

Serum Triglycerides (TG), total cholesterol (TC), and HDL-cholesterol (HDL-C) were estimated according to the methods respectively. The serum levels of VLDL and LDL cholesterol were calculated using Friedewald formula. The atherogenic index (AI) was calculated by using the following formula.

$$\text{Atherogenic index} = \frac{\text{TC} - \text{HDL-C}}{\text{HDL-C}}$$

STATISTICS

The values are expressed in mean ± SEM. One way ANOVA followed by Tukey's multiple comparison Test was used to analyse the effect of different doses of drugs when compared to control, with the help of Graph Pad Instat software, version 3.01. P.

RESULTS

Table no 7.1: List of Instruments used

Sr. No.	Name of Instrument	Description
1.	Autoanalyser	ARTOS, The versatile Autoanalyser, Sl No-SBPL/ 188/06-07
2.	Incubator	REMI Cooling Centrifuge. C-24 BL.
3.	Digital Balance	ACCULAB – Sartorious group.
4.	Flash Evaporator	SUPERFIT, Rotary "Vaccum Digital Bath", PMTc – 3040
5.	Deep Freezer	BLUE STAR, Model No.- CHE 400, Sr No.-67771
6.	Homogenizer	REMI Homogenizer Mumbai. Type – RQ 127A

Table 7.2: List of Chemicals used

S.No	Name of Chemical	Description
1.	Atorvastatin	Gifted by Microlabs Pvt. Ltd. Bangalore
2.	Heparin	(HEP-5) Gland Pharma Ltd, Hyderabad. Batch – No. UJ918
3.	Chloroform	S.D. Fine – Chem Ltd. Mumbai.
4.	KCl	S.D. Fine– Chem Ltd. Mumbai. Batch-No: -200Z-0200-1612-09. Mole. Wt:- 74.55
5.	Formalin	Fischer Scientific. Lot No:- 91026906-5., Pdt No.24005
6.	NaCl	LeoChem, Bangalore. Mole.Wt:- 58.44, Lot No. 126012
7.	EDTA	S.D.Fine Chem Ltd. Pdt No:- LO4/10204/2611/13
8.	KH_2PO_4	Qualigens fine Chemicals, Mumbai. Lot No:- 18986711-
9.	K_2HPO_4	Leochem, Bangalore. Lot No- 125176, P-2V829.
10.	H_2O_2 (30%w/v)	SDFCL-38694L05,Batch No- G09A/2209/0807/13
11.	Methanol	SDFCL. Mole Wt:- 32.04, B.P. 64-65.5 ºC, Batch No:- K08A/1308/1211/13
12.	Trichloroacetic acid	Nice Chemicals, Bombay.
13.	Thiobarbituric acid (TBA)	Loba Chemicals, Mumbai.
14.	Sodium azide	S.D. Fine Chem Ltd.
15.	Reduced glutathione	Sigma U.S.A.
19.	Urea Kit	Coral Clinical Systems, Verna Goa, India.
20.	Uric acid Kit	Coral Clinical Systems, Verna Goa, India.
21.	Creatinine Kit	Coral Clinical Systems, Verna Goa, India.

Table 7.3: Preliminary phytochemical screening of *Sapindus emarginatus* leaves extract:

S.NO.	TEST	RESULT
1.	**ALKALOIDAL TEST** a.Dragondroffs test b.Mayer's test c.Wagner's test d. Hager's test	 Positive Positive Positive Positive
2.	**CARBOHYDRATES TEST** a.Molish's test b.Fehling's test c.Benedict's test d. Baeford's test	 Positive Positive Positive Positive
3.	**STEROIDS TEST** a.LibermannBuchard test	 Positive

	b. Salwoski test	Positive
4.	**GLYCOSIDES TEST** a.Legal test b.Baljet test c.Killerkilaini test d. Borntagers test	 Positive Positive Positive Positive
5.	**SAPONINS TEST** a.Foam test	 Positive
6.	**FLAVONOIDS TEST** a.Shinoda test	 Positive
7.	**TRITERPINOIDAL TEST**	Negative
8.	**PHENOLICS & TANNINS TEST** a.Ferric chloride test b.Gelatin test c.Lead acetate test	 Negative Negative Negative
9.	**PROTIEN& AMINOACIDS TEST** a.Buret's test b.Ninhydrin test c.Xanthoprotic test	 Positive Positive Positive
10.	**FIXED OIL TEST** a.Spot test	 Positive
11.	**RESIN TEST** a.Acetic anhydride test	 Positive

Table no 7.4: Percentage yield of crude extract of *Sapindus emarginatus* leaves

S.No.	Solvent	Color and Consistency	Percentage yield
1	Methanol Leaf	Dark brown sticky	10.2%

I. Determination of Acute Oral Toxicity of EBG:

In acute toxicity study, no death was recorded, in the 14 days of observation period in the male and female animals given 2 g/kg of the Methanolic extract of *Sapindus emarginatus leaves* orally. The animals did not show any changes in the general appearance during the observation period. Based on the results, the dose was fixed by oral route for further studies.

Table no 7.5: Toxicity record sheet: The toxicity record sheet is as follows:

S.no.	Code	Toxicity		Time Of Death	Observation									
		Onset	Stop		Skin colour	Eyes	Resp	CNS	Tre	Con	Sali	Diah	Sleep	Leth
1.	MBG	x	X	x	x	X	x	X	x	x	x	x	X	x

(TRE-Tremor, CON-Convulsions, SALI- Salivation, Diah - Diarrhea, LET-Lethargy)
× = Negative Ø = Positive

II. Evaluation of Anti Hyperlipidemic activity of *Sapindus emarginatus* leaves in Rats

Effect of *Sapindus emarginatus* leaves extract on cholesterol level of rats

The hyperlipdemia control group showed significant (P<0.001) increase in cholesterol level when compared to normal control group. In hyperlipdemia control group mixed with Atorvastatin (10mg/kg); Sapindus emarginatus leaves (100, 200 mg/ kg) showed significant (P<0.001) decrease when compared to hyperlipdemia control group. The cholesterol level of MESG displayed highly appreciated decrease (P<0.001) when the values are looked upon the values of Atorvastatin (10mg/kg).

Table No 7.6: Effect of drugs on serum total cholesterol (mg/dl)

S.No.		Treatment	Cholesterol (mg/dl)
1		Normal Control	130.3 ± 13
2		HCD	201.3 ± 75 ###
3		ATORVAS 10mg/kg	146.9 ± 23 **
4		MESE 100 mg/ kg	160.1±2.32 ## ***
5		MESE 200 mg/ kg	153.2±1.27 ***

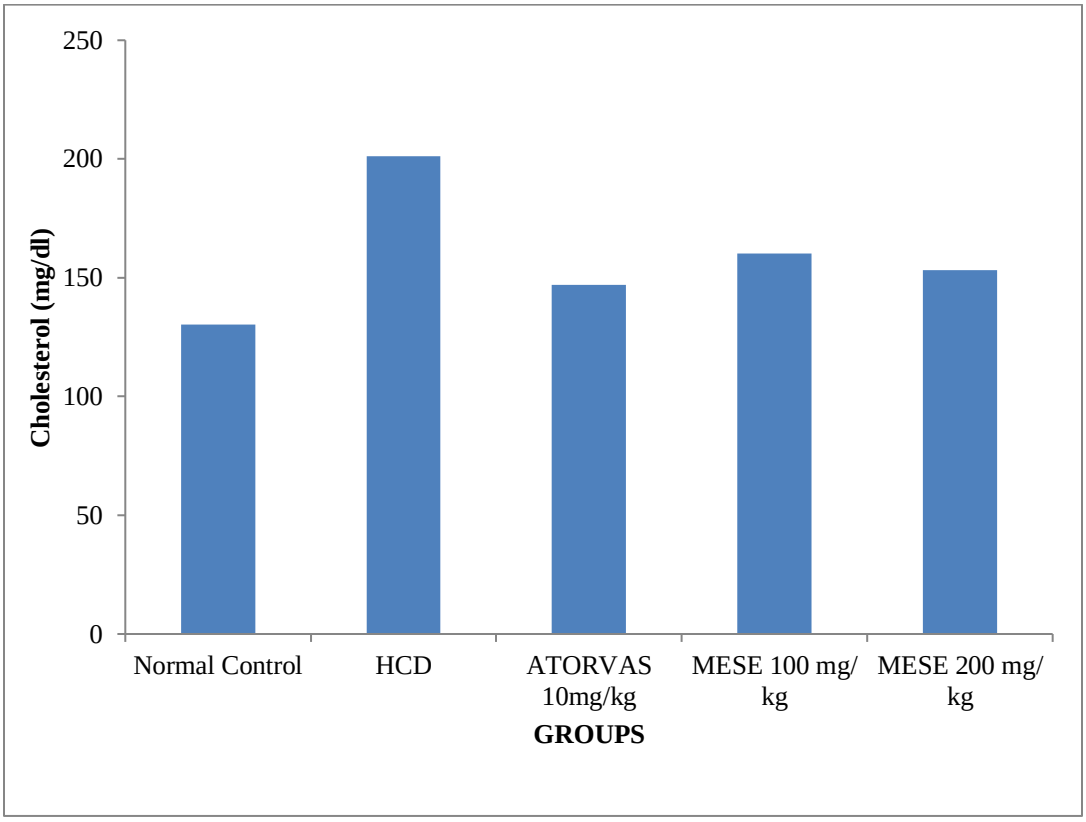

Figure 7.1: Evaluation of Total Cholesterol

Effect of Sapindus emarginatus extract on cholesterol level of rats

The values are expressed as mean ± SEM; ##P<0.01, ###P<0.001 when compared to normal control group. ***P<0.001 when compared to HCD control group. (one way ANOVA followed by Tukey's multiple comparison Test).

Effect of Sapindus emarginatus extract on HDL-C level of rats

The HDL-C levels of hyperlipidemia control group provides the reported values of the lowest order having the significant (P<0.001) decrease when those values of extract brought in comparison to normal control group. In hyperlipidemia control group stirred with Atorvastatin (10mg/kg); Sapindus emarginatus (100, 200 mg/ kg) a prominent (P<0.05 and P<0.001) increase when it related to Atorvastatin treated group.

Table No 7.7: Effect of drugs on serum HDL-C level (mg/dl)

S.No.	Treatment	HDL-C (mg/dl)
1	Normal Control	48.3 ± 0.112
2	HCD	39.36 ± 1.426 ##
3	ATORVAS 10mg/kg	46.19 ± 1.162 ***
4	MESE 100 mg/ kg	45.10 ± 1.562*
5	MESE 200 mg/ kg	47.62 ± 1.003 ***

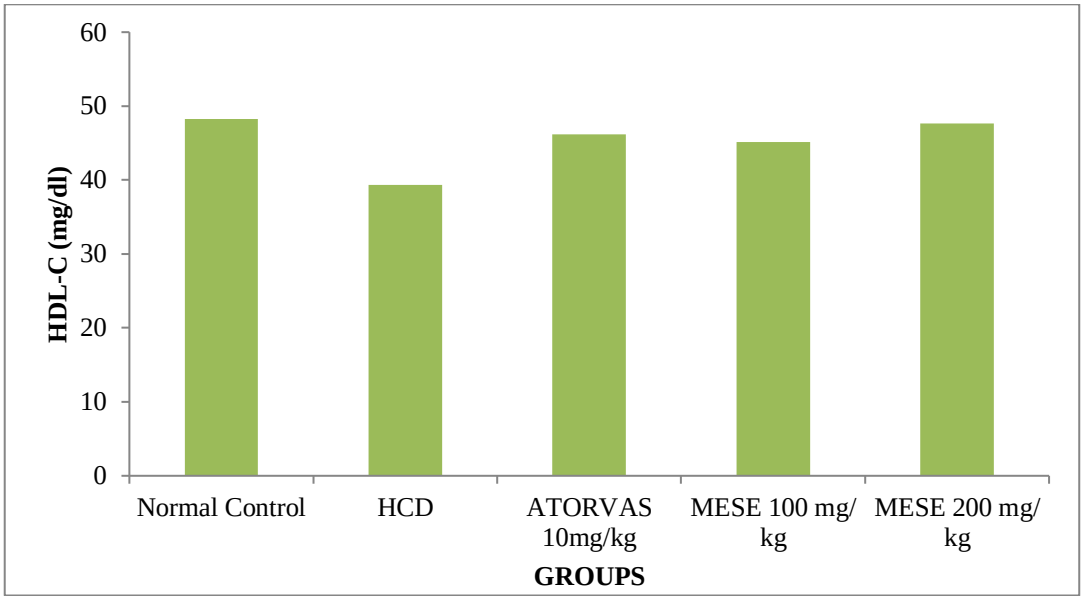

Figure 7.2: Evaluation Of serum HDL-C level

Effect of drugs on serum HDL-C level. n=6; the values are expressed as mean ± SEM; ##P<0.01, ###P<0.001, when compared to normal control group. *P<0.05, ***P<0.001 when compared to hyperlipidemia control group, P<0.05, ^P<0.001 when compared to Atorvastatin 10 mg/kg. (One way ANOVA followed by Tukey's multiple comparison Test).

Effect of Sapindus emarginatus extract on LDL-C level of rats

The HDL-C levels of hyperlipidemia control group provide the reported values of the lowest order having the significant (P<0.001) decrease when those values of extract brought in 29 comparisons to normal control group. In hyperlipidemia control group stirred with Atorvastatin (10mg/kg); Sapindus emarginatus (100, 200 mg/ kg) a prominent (P<0.05 and P<0.001) increase when it related to Atorvastatin treated group.

Table 7.8: Effect of drugs on serum LDL-C level (mg/dl)

S.No.	Treatment	LDL-C (mg/dl)
1	Normal Control	80.1±0.03
2	HCD	120.2±0.12##
3	ATORVAS 10mg/kg	73.16±0.32***
4	MESE 100 mg/ kg	90.24±0.53*
5	MESE 200 mg/ kg	84.26±0.62***

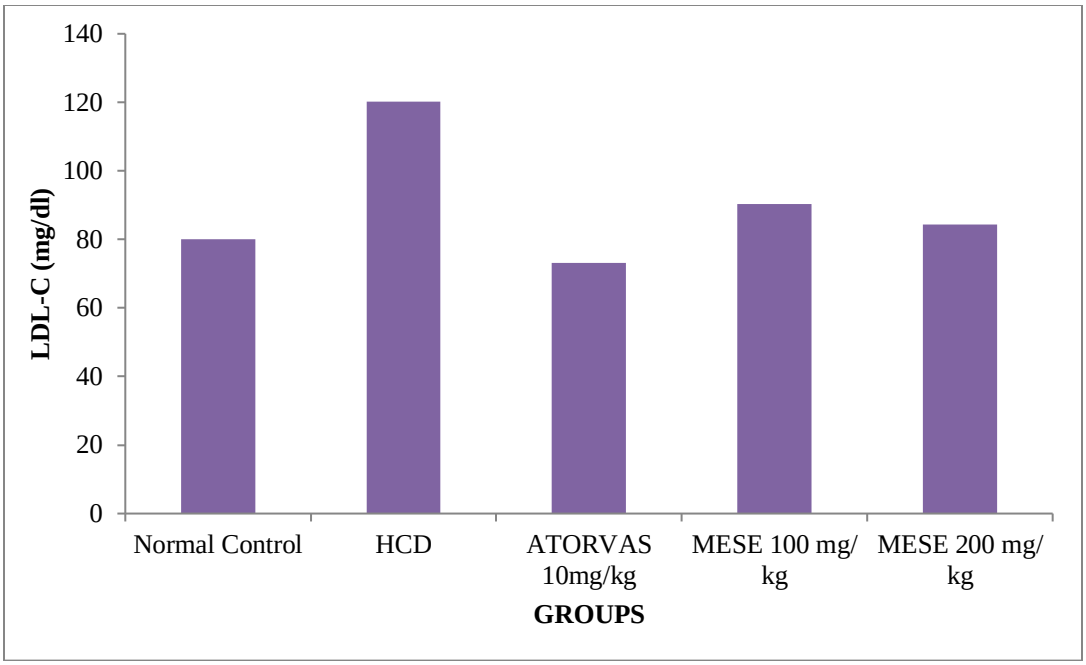

Figure 7.3: Evaluation of serum LDL-C level

Effect of drugs on serum LDL-C level. n=6; the values are expressed as mean ± SEM; ##P<0.01, ###P<0.001, when compared to normal control group. *P<0.05, ***P<0.001 when compared to hyperlipidemia control group, P<0.05, ^P<0.001 when compared to Atorvastatin 10 mg/kg. (One way ANOVA followed by Tukey's multiple comparison Test).

Effect of Sapindus emarginatus leave extract on triglyceride level of rats

The triglyceride levels of hyperlipidemia control group provides the reported values of the lowest order having the significant (P<0.001) decrease when those values of extract brought in comparison to normal control group. In hyperlipidemia control group stirred with Atorvastatin (10mg/kg); Sapindus emarginatus (100, 200 mg/ kg) a prominent (P<0.05 and P<0.001) increase when it related to Atorvastatin treated group.

Table No 7.9: Effect of drugs on serum TG level (mg/dl)

S.No.	Treatment	TG (mg/dl)
1	Normal Control	70.1±0.01
2	HCD	120.1±0.10##
3	ATORVAS 10mg/kg	86.1±0.29***
4	MESE 100 mg/ kg	100.01±0.30*
5	MESE 200 mg/ kg	96.01±0.14***

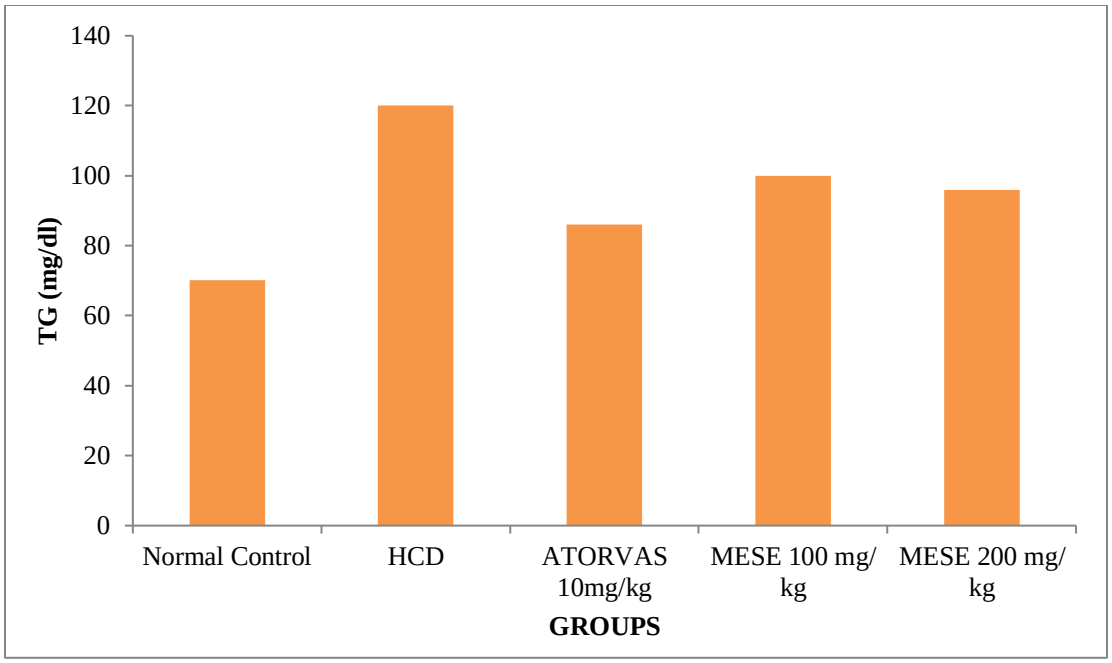

Figure 7.4: Evaluation of serum TG level

Effect of drugs on serum TG -level. n=6; the values are expressed as mean ± SEM; ##P<0.01, ###P<0.001, when compared to normal control group. *P<0.05, ***P<0.001 when compared to hyperlipidemia control group, P<0.05, ^P<0.001 when compared to Atorvastatin 10 mg/kg. (one way ANOVA followed by Tukey's multiple comparison Test).

Effect of Sapindus emarginatus leave extract on Atherogenic Index of rats.

The Atherogenic index of hyperlipidemia control group pointed out a marked rise (P<0.001) increase when the same is brought in comparison to normal control group. In hyperlipidemia control group treated with Atorvastatin (10mg/kg); MESG (100 and 200mg/kg) pointed out to a significant decrease (P<0.001) when related to hyperlipidemia control group. The atherogenic index of MESG (100 mg) reflected upon a significant (P<0.001) lowering in vales when compared with Atorvastatin 10 mg/kg.

Table 7.10: Effect of drugs on serum Atherogenic Index (mg/dl)

S.No.	Treatment	Atherogenic Index (mg/dl)
1	Normal Control	0.289 ± 0.0103
2	HCD	1.0612 ± 0.0101###
3	ATORVAS 10mg/kg	0.782 ± 0.0101***
4	MESE 100 mg/ kg	0.610 ± 0.012###**
5	MESE 200 mg/ kg	0.505 ± 0.019***

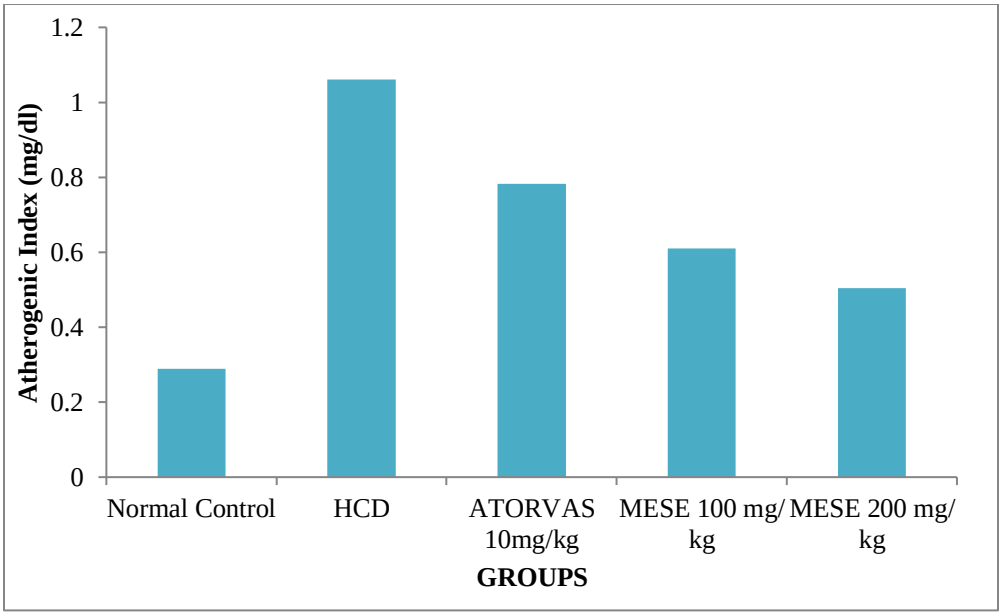

Figure 7.5: Evaluation of Atherogenic Level

DISCUSSION:

Preliminary phytochemical screenings of Methanolic extracts of whole plant of *Sapindus emarginatus* were done. Result showed the presence of the following phytochemical constituents such as total phenolic substances, glycosides, alkaloids, flavonoids, and tannins. The acute toxicity study does not show any deviation from the normal behavior. It is found that the extract is nontoxic and well tolerated. From this, LD 50 is determining from that the effective oral dose for the anti hyperlipidemic study was selected. So, the present study was aimed to evaluate the Antihyperlipidemic activity of the *Sapindus emarginatus* leave extract on HCD induced hyperlipidemia in Wistar rats. The Atorvastatin at a dose level of 10mg/kg was used as the standard drug. Hyperlipidemia was characterized by elevated serum total cholesterol (TC), low density (LDL), very low density lipoprotein (VLDL) and decrease in high density lipoprotein (HDL). These factors are exhibited to be the risk factors for coronary heart diseases. Hyperlipidemia contributes significantly in the manifestation and other development of atherosclerosis and coronary heart diseases (CHD).

Cardiovascular diseases, including atherosclerosis, are the most common cause of mortality and morbidity worldwide. The main aim of treatment in patients with hyperlipidemia is to reduce the risk of developing ischemic heart disease or the occurrence of further cardiovascular or cerebrovascular disease.

Currently available hypolipidemic drugs have been associated with a number of side effects. The consumption of synthetic drugs leads to hyperuricemia, diarrhoea, nausea, myositis, gastric irritation, flushing, dry skin and abnormal liver function Medicinal plants are used for various research purposes. Any herbal treatment for hypercholesterolemia has almost no side effects and, is relatively cheap, and locally available. They are effective in reducing the lipid levels in the system.

The *in vivo* antihyperlipidemic activity was evaluated on high cholesterol diet induced hyperlipidemic rats. High cholesterol diet, was used successfully to induced hyperlipidemia in previous studies. It causes these effects by activating HMG- CoA and inhibiting lipoprotein lipase activity). High cholesterol diet has been utilized in the hyperlipidemic model due to its convenience, reproducibility, and lack of undesirable underlying pathological conditions.

The significant reduction in serum cholesterol, TG, LDL, AI, and also increased in total HDL level in the different dose level (100,200 mg/kg) in high cholesterol diet.

Induced hyperlipidemia in rats Extract of *Sapindus emarginatus* showed (P.>;0.001) significant reduction in high cholesterol diet induced hyperlipidemia was compared with normal standard drug Atorvastatin 10mg/kg.
The hyperlipidemia control group maintained a significant (P<0.001) rise in cholesterol level when compared to normal control group. In hyperlipidemia

control group treated with Atorvastatin (10mg/kg); *Sapindus emarginatus* (100, 200 mg/ kg) displayed a significant (P<0.001) decrease when compared to hyperlipidemia control group. This indicates that methanolic extract of *Sapindus emarginatus* (MESG) significantly reduces the serum cholesterol level.

HDL is considered to be a beneficial lipoprotein as it is involved in reverse cholesterol transport and has an inhibitory effect in the pathogenesis of atherosclerosis. So increased HDL-C has a cardio protective effect. The HDL-C levels decrease in hyperlipidemia control group and significantly increase (P<0.05 and P<0.001) in Atorvastatin (10mg/kg); MESG Sapindus emarginatus (100, 200 mg/ kg).

LDL carries cholesterol from the liver to the peripheral cells and smooth muscle cells of the arteries. A rise in LDL may cause deposition of cholesterol in the arteries and aorta, a risk factor for CHD. Our study points out that there is a significant (P<0.001) increase in LDL-C level of hyperlipidemia control by comparing with normal control group and LDL-C was found to get reduced significantly (P<0.001) in Atorvastatin (10mg/kg); MESG *Sapindus emarginatus* (100, 200 mg/ kg).

The serum triglyceride levels have been reported to be an important risk factor as it influences lipid deposition and clotting mechanisms. Like cholesterol, it tends to damage vascular endothelial cells. High fat diet produces an increase in TG levels due to lipoprotein lipase triacylglycerol hydrolysis. The Triglyceride level enables a significant (P<0.001) increase in hyperlipidemia control group on comparison to normal control group. In hyperlipidemia control group blended with Atorvastatin (10mg/kg); *Sapindus emarginatus* (100, 200 mg/ kg) produced a significant (P<0.001) decrease when compared to hyperlipidemia control group. TG level of MESG 100mg, appeared to have a significant decrease when compared to Atorvastatin treated group.

Atherogenic index (AI) is predictors of coronary risk. The atherogenic index (AI) of plasma is a mathematical relationship between TG and HDL-C. The AI ranges from -0.3 to 0.1 (low), 0.1 to 0.24 (medium) and above 0.24 (high) risk of CV. The Atherogenic index of hyperlipidemia control group displayed a significant (P<0.001) increase once it is referred to normal control group. In hyperlipidemia control group treated with Atorvastatin (10mg/kg); *Sapindus emarginatus* (100, 200 mg/ kg) registered significant decrease (P<0.001) when compared to hyperlipidemia control group. The atherogenic index of MESG (100 mg) presented a significant (P<0.001) decrease when related to Atorvastatin 10 mg/kg.
MESG also decreased atherogenic index which are atherogenic coefficient, Cardiac risk ratio and Atherosclerosis index. Atherogenic index are powerful indicators of the risk of heart disease: In this study, we observed that the ERCP significantly reduced atherogenic index. According to lower atherogenic index is protective against coronary heart disease. Liver damage is always associated with cellular necrosis, increase in lipid peroxidation and depletion in the tissue GSH levels.

CONCLUSION:

Plant materials are used throughout the developed and developing world as home remedies, in over-the-counter drug products, and as raw material for the pharmaceutical industry, and they represent a substantial proportion of the global drug market. Certain herbs have become popular over the years, but the public, medical practitioners and the media still have a poor understanding of herbal medicine.

Evidence is emerging on the dangers of herbs. As in most situations, the truth lies hidden under the media hype, poorly understood science, and exaggerated claims. Lack of experience, information, and education about herbs make consumers, physicians, and other orthodox health care provider's easy victims of market exploitation and herbal myths. There is no rational reason behind the tendency to equate "natural" with "harmlessness."

The fact that something is natural does not necessarily make it safe or effective. In addition, a lack of knowledge of photochemistry leads to misinterpretation and misunderstanding. It is very likely that some herbs will have side effects, interact with other medications, and be toxic. Information on isolated constituents should not be applied directly to the whole herb and studies on in vitro forms should not be confused with oral administration which was established by pharmacological screenings. In current scenario, herbs are the potent sources of medicines used in the treatment of various disease and disorders. Since, plants are used as medicine there is prompt need of evaluation of plant species, therefore, the present work was conceived to evaluate the phytochemical and pharmacological screening of few Indian medicinal plants.

Thus the results of the present investigation clearly indicated that the selected medicinal plants possess good antihyperlipidemic activity in atherogenic diet

induced hyperlipidemic rats and led to the development of new Herbal formulation possessing antihyperlipidemic and antiatherosclerosis activities.
Hence it is going to be concluded that the potential benefits of the extracts of *Sapindus emarginatus* has been demonstrated well in advance and can be used further to demonstrate the antihyperlipidemic as well as controlling of both triglyceride levels and reducing the risk of factors of cholesterol inducers. The mentioned results of the research suggest that the *Sapindus emarginatus* found to have the potential antihyperlipidemic action.

The results found are encouraging for further studies on the selected plants and to identify the bioactive compounds.

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