International Journal of Universal Pharmacy and Bio Sciences 8(3): May-June 2019

INTERNATIONAL JOURNAL OF UNIVERSAL PHARMACY AND BIO SCIENCES

IMPACT FACTOR 4.018*

ICV 6.16*

Pharmaceutical Sciences **Research Article……!!!**

EVALUATION OF ANTI HYPERLIPIDEMIC ACTIVITY OF AQUEOUS LEAF EXTRACT OF *SYZYGIUM SAMARANGENSE* (BLUME) MERR. & L.M.PERRY ON HIGH FAT DIET FED RATS

Dr.P.AMUDHA[1*], P.SUBATHIRADEVI[2], SIJI MATHACHAN[3]

[1*] Professor, Department of Pharmacology, C.L Baid Metha College of Pharmacy, Rajiv Gandhi Salai, Old Mahabalipuram Road, Jyothinagar, Thoraipakkam, Chennai-600 097, Tamil Nadu, India.

[2] Department of Pharmacology, C.L Baid Metha College of Pharmacy, Rajiv Gandhi Salai, Old Mahabalipuram Road, Jyothinagar, Thoraipakkam, Chennai-600 097, Tamil Nadu, India.

[3] Department of Pharmacology, C.L Baid Metha College of Pharmacy, Rajiv Gandhi Salai, Old Mahabalipuram Road, Jyothinagar, Thoraipakkam, Chennai-600 097, Tamil Nadu, India.

KEYWORDS:

Syzygium samarangense Blume (merr) & L.M.Perry., hyperlipidemia, Organization for economic corporation and development(OECD), ALESS (Aqueous extract of *Syzygium samarangense* (Blume) Merr & L.M.Perry, Histopathology, Anti-hyperlipidemic drugs, HFD.

FOR CORRESPONDENCE:
Dr.P.Amudha *
ADDRESS:
Professor, Department of Pharmacology, C.L Baid Metha College of Pharmacy, Rajiv Gandhi Salai, Old Mahabalipuram Road, Jyothinagar, Thoraipakkam, Chennai – 600 097, Tamil Nadu, India.

ABSTRACT

Hyperlipidemia is an elevation of lipids (fats) in the blood stream. These lipids includes cholesterol, cholesterol esters, phospholipids and triglycerides. They are transported in blood in the form of large molecules called lipoproteins. Hyperlipidemia usually has no noticeable symptoms but however deposits of cholesterol known as xanthomas may form under the skin especially under the eyes or around the Achilles tendon in individual with the familial forms of disorder or in those with very high levels of cholesterol in the blood. Individuals with hypertriglyceridemia may develop numerous pimples like lesions across the body. Higher levels are associated with lipemiaretinalis (white appearance of the retina) eruptive xanthomas (small lumps in the skin, sometimes itchy). Examples of Antihyperlipidemic drugs are HMG CO-A reductase, Fibrate, bile acid sequestrants, ezetimibe, nicotinic acids. Many plants were used as anti-hyperlipidemic drug in herbal medicines. The *Syzygium samarangense* (Blume) Merr & L.M.Perry belongs to family Myrtaceae. The evaluation of anti-hyperlipidemic activity of *Syzygium samarangense* (Blume) Merr&L.M.Perry performed by following procedure. ALESS was subjected to preliminary phytochemical screening, acute oral toxicity and antihyperlipidemic study. The acute oral toxicity was performed as per OECD 423 guidelines. The animals were divided into 5 groups each constituting 6 rats. Group I were normal rats, Group II were induced HFD. Group III animals were treated with simvastatin 10mg/kg b.w/p.o. Group IV, V were treated with ALESS 200mg/kg, 400mg/kg b.w/p.o. for 14 days. On 31st day animals were sacrificed and biochemical estimation were performed. Histopathology of liver and adipose tissue were carried out.

INTRODUCTION:

High cholesterol complication begins with atherosclerosis; atherosclerosis is the buildup of fatty deposits on the inner side of arteries called as plaque. If plaque starts build up in an artery, the artery gradually become narrows. Due to narrowed artery, the blood flow is reduced and thus nutrients and oxygen shortage in the brain, heart and other parts of the body is common, The artery may also become less elastic and causes hardening of the arteries [1].

Hyperlipidemia is an elevation of lipids (fats) in the blood stream. These lipids includes cholesterol, cholesterolesters, phospholipids and triglycerides. They are transported in blood in the form of large molecules called lipoproteins [2]. Prognosis of hyperlipidemia is one of the important risks causing coronary heart disease, Hyperlipidemia is closely related to cardiovascular disease, obesity and easy to cause stroke, hypertension, gallstones, pancreatitis, hepatitis and lead to male sexual dysfunction and Alzheimer's disease. New researches show that hyperlipidemia relates with the incidence of cancer [3].

The World Health Organisation estimates that 80% of the world's population presently uses herbal medicine for some aspect of primary health care [4].

Hypercholestremia in which there is a high level of cholesterol and Hypertriglyceridemia, in which there is a high level of triglycerides, the most common form of fat. Herbal medicine is a major component in all traditional medicine systems and a common element in siddha, ayurvedic, homeopathic, naturopathic, traditional Chinese medicine, and native American medicine, Herbs can help the patient resist disease, and that in addition, they provide nutritional and immunological support that pharmaceuticals lack [5]. In the present study, antihyperlipidemic activity of aqueous leaf extracts of *Syzygium samarangense* have been evaluated.

MATERIALS AND METHODS:

Preparation of Plant Extracts:

The leaves of *Syzygyium samarangense* (Blume) Merr.&L.M.Perry collected from Kerala, South India in the month of august 2016 and authenticated by Dr.SasikalaEthirajulu, Research officer, Pharmacognosy, Central Research institute for Siddha, Chennai-106.

The leaves of *Sygyzium samarangense* (Blume) Merr.&L.M.Perry were shade dried and pulverised using a standard pulveriser. A weighed quantity of powdered leaves were subjected to continous hot extraction in soxhlet apparatus with water at 60- 80^{O}C. The filtered extract was evaporated under reduced pressure using rotary vaccum evaporator until all solvent was removed to give a dark coloured molten extract. The aqueous extract of *Syzygium samarangense* (Blume) Merr.&L.M.Perry was used for the study(ALESS).

Acute Oral Toxicity Study:

The Acute Oral Toxicity Study was done according to the OECD guidelines 423. A single administration of 2000 mg/kg b.w/p.o of the ALESS for three days in 3 female wistar rats and observed for 14 days. Body weights of rats before and after administration were observed for morbidity and mortality. Any changes in skin, fur, eyes, mucous membrane, respiratory, circulatory, autonomic & central nervous system, motor activity and behaviour pattern were observed and also sign of tremors, convulsion, salivation, diarrhoea, lethargy, sleep and coma were noted [6].

Experimental Animals:

Wistar rats of weighing 150-200 gm were used for this study. They were housed five per cage under standard laboratory conditions at a room temperature at 22 ± 2^0 C with 12 hr light/dark cycle for 7 days. The animals were acclimatized to laboratory conditions one week and provided with standard pellet chow with water *ad libitum*. Ethical committee approval was obtained from IAEC of C.L. Baid Metha College of Pharmacy. The approval number is **IAEC/XLIX/04/CLBMCP/2016.**

High Fat Diet Induced Hyperlipidemia:

Composition of HFD:

The chronic experimental Hyperlipidemia was produced by feeding high fat diet. The high fat diet contains following ingredients per 100gm of basal rodent chow: **casein 30gm, agar 2gm, cellulose 8gm, sucrose 51gm, vitamin mixture 0.5gm, mineral mixture 0.5gm, cholesterol 1.5gm, coconut oil 8ml.**

Cholesterol 1.5gm was suspended in 8 ml of coconut oil and mixed with 100gm of rodent chow along with other ingredients of the high fat diet. Pellets were made shade dried and used as high fat diet to induce Hyperlipidemia. The HFD was followed for 30 days [7].

Animal Grouping:

Thirty Wistar rats weighing 150 to 250 gm were randomly divided into five groups of six each and kept in polypropylene cages for 5 days prior dosing for acclimatization to the laboratory conditions. The drugs were administered in constant volume of 0.2ml/100gm body weight. The control animals received the vehicle in the same volume p.o.

Experimental Procedure:

Table 1:

GROUPS	TREATMENT
Group I	Fed with standard rodent chow and served as normal saline from day 0
Group II	Fed with high fat diet served as negative control for 30 days
Group III	Fed with HFD and simvastatin 10mg/kg/p.o for 30 days
Group IV	Fed with HFD and lower doses (LD) of ALESS (200mg/kg/p.o) for 30 days
Group V	Fed with HFD and higher doses (HD) of ALESS (400mg/kgp.o) for 30 days

On day 31st animals were anaesthetized with chloroform and blood was collected by decapitation. The blood was subjected to centrifugation to obtain serum. Serum was analyzed for serum TGs, Serum TC, Serum HDL, Serum LDL, Serum VLDL. Liver was analyzed for liver TGs, liver TC, Total protein. Histopathological analysis of liver and adipose tissue was done.

RESULTS:

PRELIMINARY PHYTOCHEMICAL ANALYSIS OF AQUEOUS LEAF EXTRACT OF *Syzygium samarangense* (Blume) Merr. &L.M.Perry

The result of preliminary phytochemical analysis of ALESS shows Presence of Alkaloids, Carbohydrates, Proteins, Sterols, Phenols, Flavanoids, Terpenes, Tannins and Absence of Saponins and Gums and mucilage [8]. (Table 1)

ACUTE ORAL TOXICITY STUDIES:

There was no considerable change in body weight before and after treatment and no sign of toxicity was observed. (Table 2)

HIGH FAT DIET INDUCED HYPERLIPIDEMIA:

Effect of ALESS on Serum Triglyceride Level in Hyperlipidemia Induced Model:

There was significant increase in serum TG in Group II ($P<0.001$), Group III ($P<0.01$), Group IV ($P<0.001$), Group V ($P<0.01$) when compared to Group I. There was significant decrease in serum TG in Group III, IV, V ($P<0.001$) when compared to Group II. (Table 3, Figure 1)

Effect of ALESS on Serum Total Cholesterol (Serum TC) Levels in Hyperlipidemia Induced Model:

There was significant increase in serum TC in Group II, IV ($P<0.001$), Group III, V ($P<0.01$) when compared to Group I. There was significant decrease in serum TC in Group III, IV, V ($P<0.001$) when compared to Group II. (Table 3, Figure 2)

Effect of ALESS on Serum HDL Level in Hyperlipidemia Induced Model:

There was significant decrease in serum HDL in Group II, III, IV, V ($P<0.001$) when compared to Group I. There was significant increase in serum HDL in Group III, V ($P<0.001$), Group IV ($P<0.01$) when compared to Group II. (Table 3, Figure 3)

Effect of ALESS on Serum LDL Level in Hyperlipidemia Induced Model:

There was significant increase in serum LDL in Group II, IV ($P<0.001$), Group V ($P<0.01$) when compared to Group I. There was significant decrease in serum LDL in Group III ($P<0.01$) when compared to Group I. There was significant decrease in serum LDL in Group III ($P<0.001$), Group IV ($P<0.05$), Group V ($P<0.001$) when compared to Group II. (Table 3, Figure 4)

Effect of ALESS on Serum VLDL Level in Hyperlipidemia Induced Model:

There was significant increase in serum VLDL in Group II, IV (P<0.001), Group III, V (P<0.01) when compared to Group I. There was significant decrease in serum VLDL in Group III, V (P<0.01), Group IV (P<0.05) when compared to Group II. (Table 3, Figure 5)

Effect of ALESS on Serum HDL/LDL Ratio in Hyperlipidemia Induced Model:

There was significant decrease in serum HDL/LDL ratio in Group II (P<0.01), Group III, IV, V (P<0.001) when compared to Group I. There was significant increase in serum HDL/LDL ratio in Group III, V (P<0.01), Group IV (P<0.05) when compared to Group II. (Table 3, Figure 6)

Effect of ALESS on Atherogenic Index (AI) in Hyperlipidemia Induced Model:

There was significant increase in AI in Group II, IV (P<0.001), Group III, V (P<0.01) when compared to Group I. There was significant decrease in AI in Group III, V (P<0.01), Group IV (P<0.05) when compared to Group II. (Table 3, Figure 7)

Effect of ALESS on Serum Glutamate Oxaloacetate Transaminase (SGOT) in Hyperlipidemia Induced Model:

There was significant increase in SGOT in Group II, IV (P<0.001), Group III, V (P<0.01), when compared to Group I. There was significant decrease in SGOT in Group III, V (P<0.001), Group IV (P<0.05) when compared to Group II. (Table 4, Figure 8)

Effect of ALESS on Serum Glutamate Pyruvate Transaminase (SGPT) in Hyperlipidemia Induced Model:

There was significant increase in SGPT in Group II, IV (P<0.001), Group III, V (P<0.01), when compared to Group I. There was significant decrease in SGPT in Group III, V (P<0.001), Group IV (P<0.01) when compared to Group II. (Table 4, Figure 9)

Effect of ALESS on Liver Triglyceride (Liver TG) Level in Hyperlipidemia Induced Model:

There was significant increase in liver TG in Group II, III, IV (P<0.001), Group V (P<0.01) when compared to Group I. There was significant decrease in liver TG in Group III, IV, V (P<0.05) when to Group II. (Table 4, Figure 10)

Effect of ALESS on Liver Cholesterol (Liver TC) Levels in Hyperlipidemia Induced Model:

There was significant increase in liver TC in Group II, III, V (P<0.01), Group IV (P<0.001) when compared to Group I. There was significant decrease in liver TC in Group III, IV, V (P<0.01) when compared to Group II. (Table 4, Figure 11).

Effect of ALESS on Liver Total Protein Levels in Hyperlipidemia Induced Induced Model:

There was significant increase in liver total protein in Group II, IV (P<0.001), Group III, V (P<0.01) when compared to Group I. There was significant decrease in liver total protein in Group III, V

(P<0.05), Group IV (P<0.01) when compared to Group II. (Table 4, Figure 12).

Effect of ALESS on Body Weight on 7th day in Hyperlipidemia Induced Model:

There was significant increase in body weight in Group II (P<0.001), Group III, V (P<0.05), Group IV (P<0.01) when compared to Group I. There was significant decrease in Group III, V (P<0.05), Group IV (P<0.01) when compared with Group II. (Table 5, Figure 13)

Effect of ALESS on Body Weight on 14th day in Hyperlipidemia Induced Model:

There was significant increase in body weight in Group II (P<0.001), Group IV (P<0.01) when compared to Group I. There was significant decrease in body weight in Group III (P<0.05), Group V (P<0.05) when compared to Group I. There was significant decrease in body weight in Group III, V (P<0.05), Group IV (P<0.01) when compared to Group II. (Table 5, Figure14)

Effect of ALESS on Body Weight on 28th day in Hyperlipidemia Induced Model:

There was significant increase in body weight in Group II (P<0.001) when compared to Group I. There was significant decrease in Group III, V (P<0.05), Group IV (P<0.01) when compared to Group I. There was significant decrease in body weight in Group III, V (P<0.05), Group IV (P<0.01) when compared to Group II. (Table 5, Figure 15)

Table: 2 Preliminary Phytochemical analysis of aqueous leaf extract of *Syzygium samarangense* (Blume) Merr. & L.M.Perry

S.no.	Constituents	Absence/presence
1.	Carbohydrates, Alkaloids, Phenols, Flavonoids, Tannins, Terpene, Sterols, Proteins	Presence
2.	Saponins, Gums and Mucilage	Absence

High Fat Diet Induced Hyperlipidemia:**Table 3:**

GROUPS	Serum Triglyceride mg/dl	Serum total cholesterol mg/dl	Serum HDL level mg/dl	Serum LDL level mg/dl	Serum VLDL level mg/dl	Serum HDL/LDL ratio mg/dl	Atherogenic index
GROUP I	58.50±1.17	69.17±1.19	49.00±1.36	51.17±1.35	12.83±1.01	1.26±0.05	1.31±0.01
GROUP II	195.50±1.40 a ***	103.00±2.06 a ***	31.17±0.94 a ***	74.33±1.38 a ***	37.83±1.24 a ***	0.51±0.01 a **	3.56±0.02 a ***
GROUP III	136.30±1.22 a **b ***	88.00±2.06 a **b***	39.00±1.33 a ***b***	50.00±1.86 a **b***	23.50±0.99 a **b**	0.65±0.01 a ***b**	1.32±0.01 a **b**
GROUP IV	185.00±1.06 a ***b***	98.00±2.47 a ***b***	41.33±1.49 a ***b**	67.17±1.62 a ***b*	36.00±2.04 a ***b*	0.54±0.01 a ***b*	2.55±0.02 a ***b*
GROUP V	104.30±1.35 a **b ***	72.30±1.68 a **b***	43.67±1.72 a ***b***	51.67±2.69 a **b***	22.00±0.99 a **b**	0.60±0.01 a ***b**	1.60±0.02 a **b**

Effect on Body Weight:

Table4:

GROUPS	Body weight (gms) on 7th day	Body weight (gms) on 14th day	Body weight (gms) on 28th day
GROUP I	138.3±3.80	142.7±3.93	149.2±4.16
GROUP II	151.7±3.80 a ***	156.3±3.08 a ***	155.8±3.96 a ***
GROUP III	141.7±3.33 a *b*	140.5±4.64 a *b*	143.3±2.47 a *b*
GROUP IV	146.7±4.41 a **b**	143±2.30 a **b**	144.0±3.00 a **b**
GROUP V	140.8±3.0 a *b*	142.5±3.58 a *b*	145.0±2.38 a *b*

The values are expressed as mean ± SEM of 6 animals

Comparisons were made between:

- ✓ Group I vs Group II, III, IV,V
- ✓ Group II vs Group III, IV, V

Statistical significance test for comparison was done by One way ANOVA followed by Dunnett's test.

Figure: 1

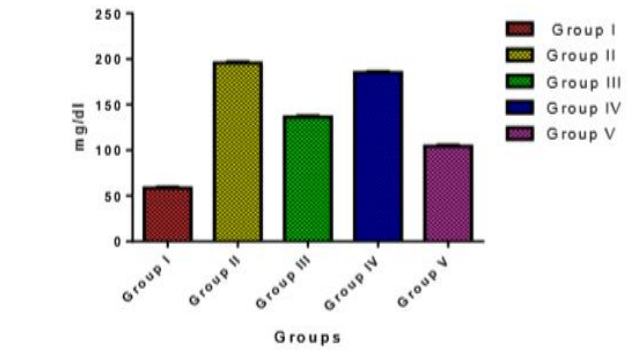


Figure:2

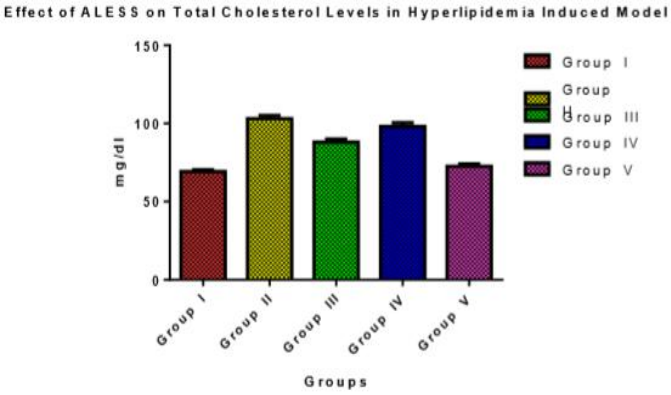


Figure: 3

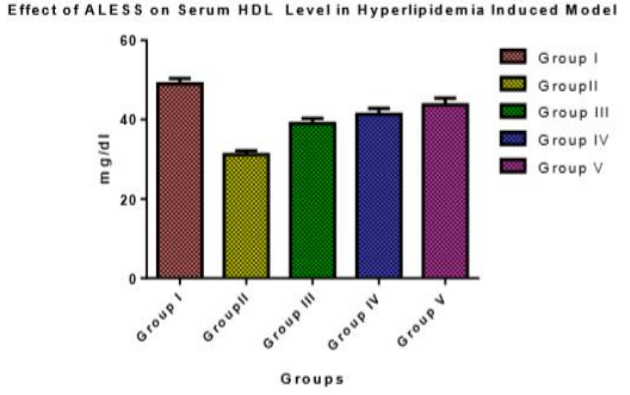
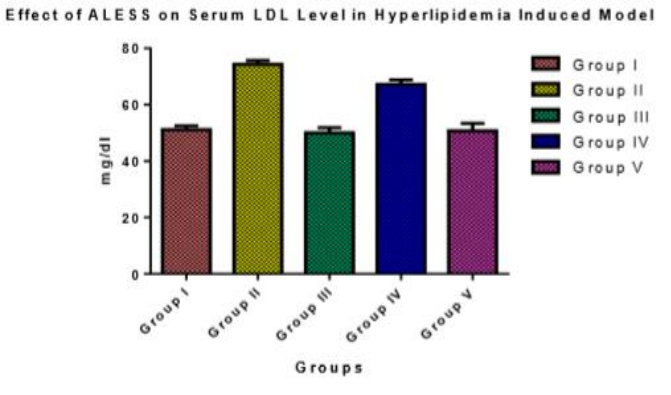


Figure: 4



Symbols represent statistical significance * p<0.05, ** p<0.01, *** p<0.001, ns –non significant

Figure: 5

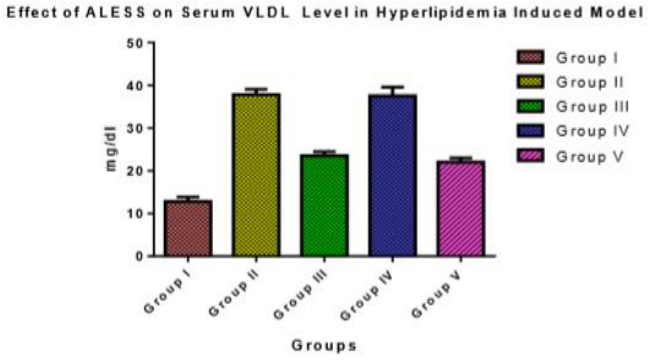


Figure:6

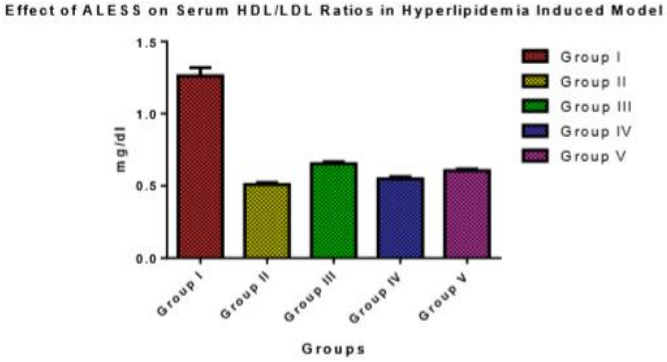


Figure: 7

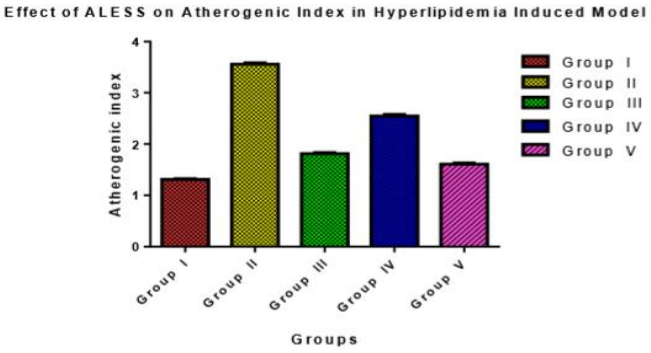


Figure: 8

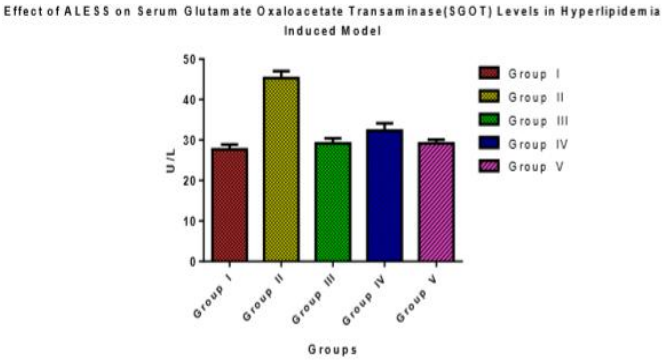


Figure: 9

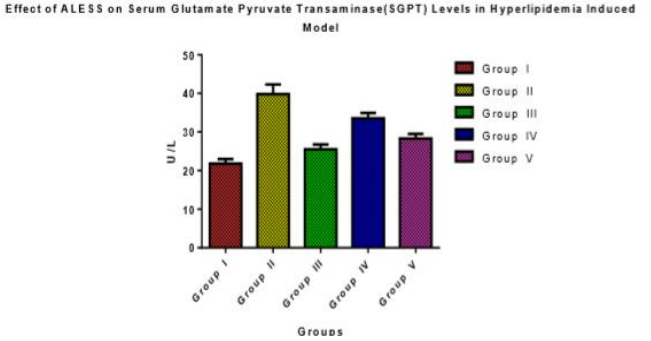


Figure: 10

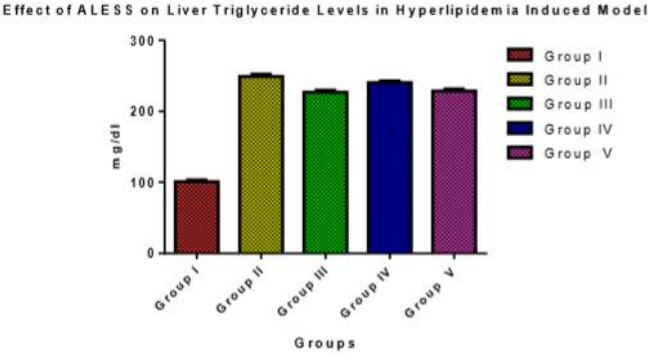


Figure: 11

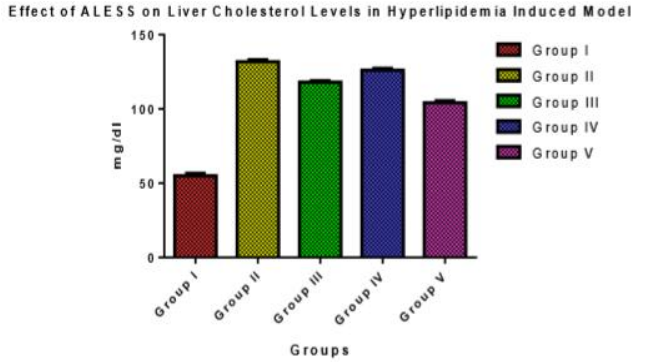
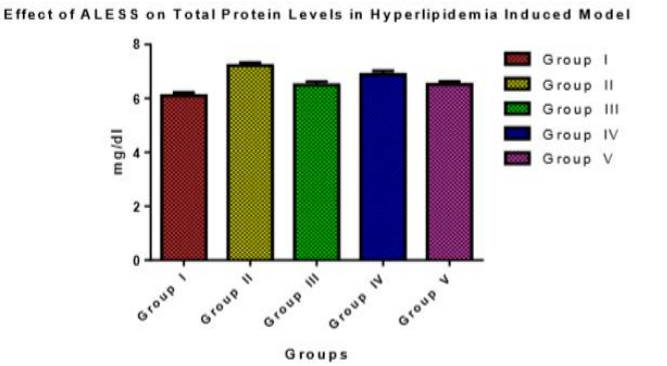


Figure: 12



 International Standard Serial Number (ISSN): 2319-8141

Figure: 13

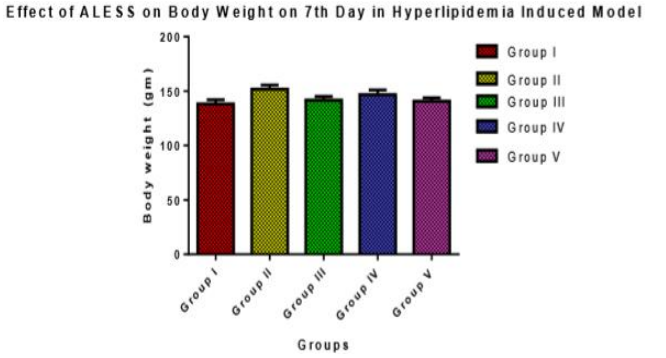


Figure: 14

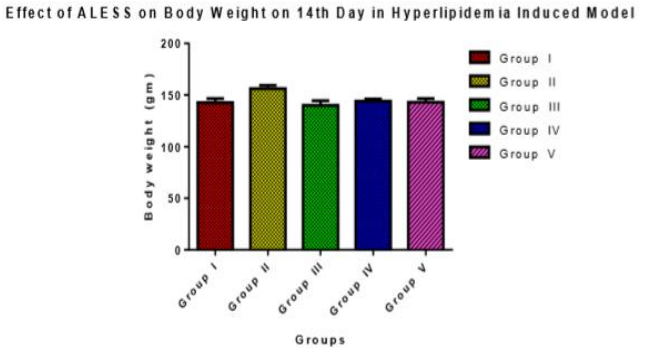


Figure: 15

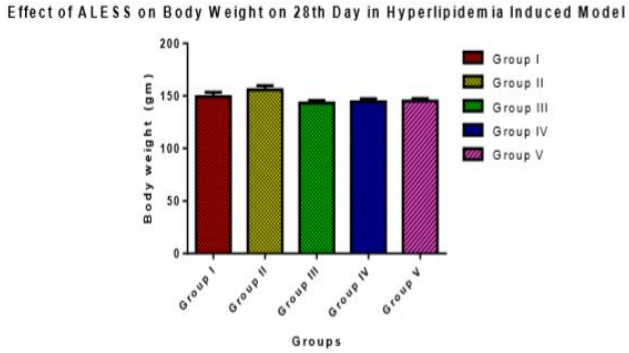
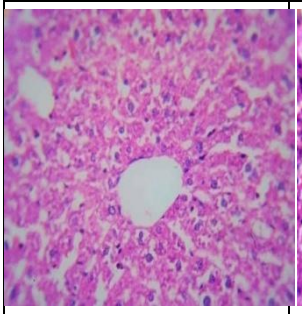
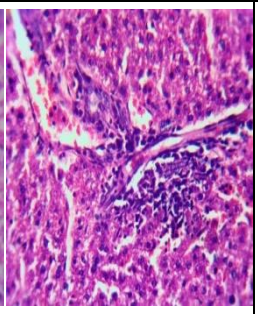
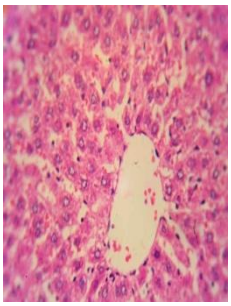
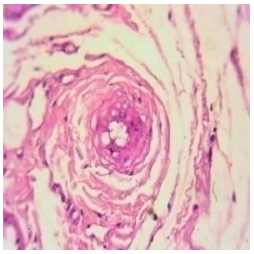
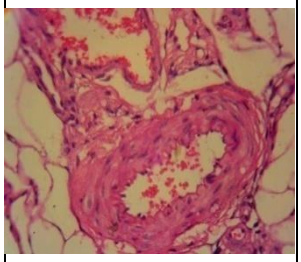
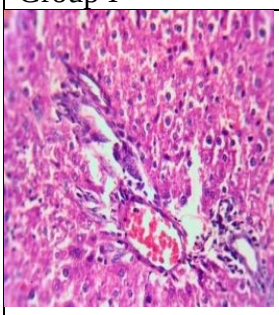
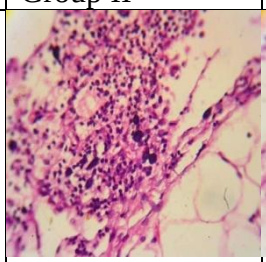
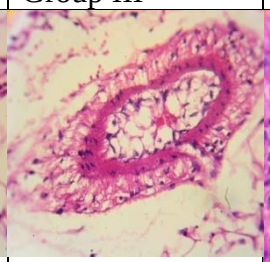
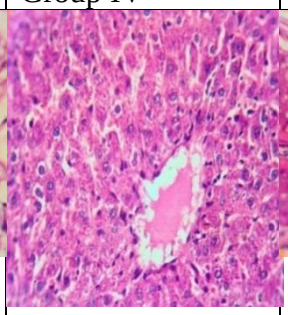
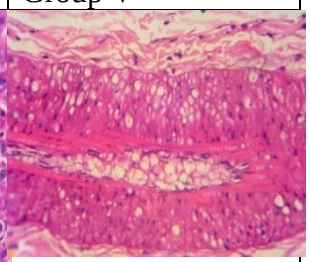


Figure: 17 Histopathology of Liver

Group I	Group II	Group III	Group IV	Group V
				

Histopathology of Adipose tissue:

Group I	Group II	Group III	Group IV	Group V
				

DISCUSSION:

Cholesterol is an essential lipid for mammalian life, but a high cholesterol level can almost guarantee the eventual onset of vascular disease and in some cases, can lead to death. It is been shown that there is a direct connection between high cholesterol level and vascular disease [9]. Cholesterol feeding has been often used to elevate serum or tissue cholesterol to assess the Hyperlipidemia related metabolic disturbances in animal [10]. It is assumed that a high level of saturated fat in addition to cholesterol is required to significantly elevate serum TG level in rat model [11].

In the present study, the ALESS showed a significant reduction in the serum cholesterol levels in HFD induced Hyperlipidemic model of rats, which was almost comparable to that of the standard drug simvastatin used in treatment. Their actions may be due to increased inhibition of intestinal absorption of cholesterol, interference with lipoprotein, increased expression of hepatic LDL receptors and their protection leading to an increased degradation and catabolism of cholesterol from the body.

Serum triglycerides are transported mainly in chylomicrons and VLDL which contains cholesterol in lower quantities, a large increase of cholesterol in plasma may unavoidably enhance the production of both the lipoprotein fractions, resulting in an increased level of their components including triglycerides in circulation. HFD fed groups treated with ALESS showed significant lowering effect on serum triglyceride levels. The serum triglyceride reducing activity of ALESS might be related to decreased absorption as well as increased excretion of these lipids via feces. The other postulated factors include reduced VLDL secretion by the liver, repressed hepatic lipogenesis or accelerated clearance by peripheral tissues.

The biochemical estimations shown that the ALESS increased the protective HDL level. The possible mechanism of ALESS may involve increase of HDL-C which is attributed to the mobilisation of cholesterol from peripheral cells to the liver by the action of Lecithin Cholesterol O-acyltransferrase [12]. The increased HDL-C facilitates the transport of TG or cholesterol from serum to liver by a pathway termed “reverse cholesterol transport” where it is catabolised and excreted out of the body [13] and might serve as a cardio protective factor to prevent the gradual initiation of atherosclerotic process.

High fat diet feeding raised the LDL and VLDL cholesterol. The increase in the concentration of LDL and VLDL observed are mainly due to the dietary carbohydrates and cholesterol in the HFD [14]. ALESS showed a significant reduction in both LDL and VLDL levels of groups fed with HFD for 30 days. The possible mechanism may be due to the expression of hepatic LDL receptor and by reverse cholesterol transport.

The LDL and HDL ratios are considered better prediction for the prevention of CHD. These levels in the study showed marked increase with LD and HD of ALESS.

In the study, HFD fed rats showed increase in atherogenic index (AI). Increased levels of TC, LDL and TGs as well as low HDL levels are responsible for the high value of AI. Administration of ALESS to HFD fed groups significantly reduced AI, which may be in relation to decreased levels of TC, LDL and TGs [15].

A marked hike is observed in the activities of SGOT and SGPT in serum of HFD fed rats indicates injury caused to liver and heart tissues due to the Hyperlipidemia. When cell membrane gets damaged, these enzymes which are normally located in the cytosol leak into the blood stream thus manifesting damage effected to liver and heart tissues [16]. The attainment of near normalcy levels of SGOT and SGPT in HFD fed gropus treated with ALESS unravels the hepatoprotective effect of ALESS in HFD induced hyperlipidemic condition.

There was an increase in hepatic cholesterol and triglyceride levels in HFD fed rats. This can be supported by evidence that feeding diet supplemented with cholesterol and cholic acid markedly increased liver weight, hepatic triglycerides and cholesterol in geese [17]. Inclusion of saturated fatty acids in the diet has been shown to produce Hyperlipidemia effect in rats. It is possible that the normal catabolism of liver lipids was impaired in the rats fed with HFD diet with consequent accumulation of lipids in the liver. HFD diet fed groups treated with ALESS showed a significant reduction in the liver cholesterol and triglycerides along with HFD diet. The possible mechanism could be the normal attainment of lipid catabolism by the liver [18].

Based on the data, the body weight of HFD diet fed rats increased up to 30^{th} day of the feeding. This is probably due to supplementation with cholesterol and fat and carbohydrates, ALESS effectively prevented the increase in body weight to a significant extend.

The histopathological observations of rats liver and adipose tissue showed that, in liver HFD group showed degeneration and fatty infiltration in hepatocytes. However HFD fed group treated with ALESS showed mild to moderate reduction in the granular degeneration and fatty infiltration. In the adipose tissue, HFD group increased deposition of adipocytes, Whereas HFD group fed with ALESS showed mild to moderate reduction in the deposition of adipocytes.

In the present study the metabolism of High fat diet rats with ALESS, showed significant difference from that of the standard drug in the following biochemical profiles Total cholesterol, HDL, Triglycerides.

These observations are clinical significance in the management of hypercholestremia with plant drug, with respect to dosage adjustment for longer duration of treatment, the benefits of ALESS in the management of cholesterol mechanism in High fat diet rats may be attributed to its better bioavailability over the standard drug.

CONCLUSION:

An increase in HDL and decrease in TC, LDL and triglycerides is associated with a decrease in the risk of ischaemic heart diseases. Most of the antihyperlipidemic drugs are causing significant reduction in both TC and HDL cholesterol levels. In the present study, significant decrease of cholesterol in the ALESS treated groups is manifested in all the lipoprotein fractions.

In conclusion, the findings of the study suggest that administration of ALESS(200/400mg/kg.,p.o) to hypercholestremia induced animals resulted in decrease of TC,LDL ,TG ,VLDL,AI and increase in HDL levels. Liver enzymes such as SGOT and SGPT are considered to be biochemical markers for assessing liver function. Hepatoxicity is evidenced by an elevation of serum marker enzymes. In this study the ALESS prevented the elevation of SGOT and SGPT.

On comparison of simvastatin with ALESS produced significantly higher HDL cholesterol ratio and lower total cholesterol and triglycerides parameters.

This study further extends the scope for clinical efficacy study of ALESS in human subjects for its better bioavailability and less loading of drugs to avoid side effects.

ACKNOWLEDGEMENT

We are very thank full to the Department of Pharmacology, Principal and management of C. L. Baid Metha College of Pharmacy for providing the facilities to conduct the result.

REFERENCES:

1. Shoshana R, Nissim G. The activity and absorption relationship of cholesterol and phytosterols. Colloids and Surfaces A Physiochemical and Engineering aspects 2006; 282:435-456.
2. American heart association. Prevention and Treatment of High Cholesterol (Hyperlipidemia). Available from URL https://www.heart.org/en/health-topics/cholesterol/prevention-and-treatment-of-high-cholesterol-hyperlipidemia.
3. Fortmann SP, Ford E, Criqui MH, Folsom AR, Harris TB, Hong Y, Pearson TA, Siscovick D, Vinicor F, Wilson PF. CDC/AHA workshop on markers of inflammation and cardiovascular disease: application to clinical and public health practice: report from the population science discussion group. Circulation. 2004 Dec 21;110(25):e554-9.
4. Kamboj VP. Herbal medicine. Current science. 2000 Jan 10;78(1):35-9.
5. Grassmann J, Hippeli S, Elstner EF. Plant's defence and its benefits for animals and medicine: role of phenolics and terpenoids in avoiding oxygen stress. Plant Physiology and Biochemistry. 2002 Jun 1;40(6-8):471-8.
6. Ecobichon DJ. The basis of toxicology testing. CRC press. Newyork.

7. Bahramikia S, Yazdanparast R. Effect of hydroalcoholic extracts of Nasturtium officinale leaves on lipid profile in high-fat diet rats. Journal of ethnopharmacology. 2008 Jan 4;115(1):116-21.
8. Kokate Ck, Purohit AP, Gokhale SB. Text book of Pharmacognosy. Nirali prakashan. 2004;108-109.
9. Dawber TR, Moore FE and Mann GV. Coronary heart disease in the Framingham study. American Journal of Public Health 1957;47:1.
10. Hozumi T, Yoshida M, Ishida Y, Mimoto H, Sawa J, Doi K, Kazumi T. Long-term effects of dietary fiber supplementation on serum glucose and lipoprotein levels in diabetic rats fed a high cholesterol diet. Endocrine journal. 1995;42(2):187-92.
11. Watts GF, Jackson P, Mandalia S, Brunt JN, Lewis ES, Coltart DJ, Lewis B. Nutrient intake and progression of coronary artery disease. The American journal of cardiology. 1994 Feb 15;73(5):328-32.
12. Khanna AK, Rizvi F, Chander R. Lipid lowering activity of Phyllanthus niruri in hyperlipemic rats. Journal of ethnopharmacology. 2002 Sep 1;82(1):19-22.
13. Jayasooriya AP, Sakono M, Yukizaki C, Kawano M, Yamamoto K, Fukuda N. Effects of Momordica charantia powder on serum glucose levels and various lipid parameters in rats fed with cholesterol-free and cholesterol-enriched diets. Journal of ethnopharmacology. 2000 Sep 1;72(1-2):331-6.
14. Sandhya T and Dhar SC. Effect of a new herbo – mineral hypolipidemic agent on plasma lipoprotein pattern in rat atherosclerosis. Indian Journal of Experimental Biology 1990;28:657-60.
15. Dabhi JK, Solanka JK, Anita M. Atherosclerotic activity of ibuprofen, a non-selective COX inhibitor – an animal study. Indian Journal of Experimental Biology 2008;46:476-81.
16. Wasan KM, Najafi S, Wong J, Kwong M, Pritchard PH. Assessing plasma lipid levels, body weight, and hepatic and renal toxicity following chronic oral administration of a water soluble phytostanol compound, FM-VP4, to gerbils. J Pharm Pharm Sci. 2001 Sep 1;4(3):228-34.
17. Eder K. The effect of a combined dietary treatment with cholesterol and cholic acid on these lipid metabolism of geese at low or high Choline concentrations. Journal of Animal Physiology and Animal Nutrition 1999;52:285-97.
18. Brai BI, Odetola AA, Agomo PU. Hypoglycemic and hypocholesterolemic potential of Persea americana leaf extracts. Journal of medicinal food. 2007 Jun 1;10(2):356-60.