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EVALUATION OF ANTIHYPERLIPIDEMIC ACTIVITY OF METHANOLIC LEAVES EXTRACT OF *LASIA SPINOSA* AND ITS ROLE IN PREVENTION OF HYPERLIPIDEMIA INDUCED PANCREATITIS IN RATS

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ABSTRACT: Leaves, rootstock, fruit and decoction of Lasia spinosa (Araceae) are used in folklore medicine for Gastrointestinal, Respiratory and Skin diseases. The intent of the present study was to evaluate the Antihyperlipidemic activity of methanolic leaves extract of *Lasia spinosa* and for its role in the prevention of hyperlipidemia induced pancreatitis in rats. The evaluation of the Antihyperlipidemic activity and the role of *Lasia spinosa* in the prevention of hyperlipidemia induced pancreatitis were done by means of Cholesterol 100 mg/kg body weight, p.o, induced hyperlipidemic model and Triton-X 100 (480 mg/kg, b.w; i.p.) induced hyperlipidemic model. The biochemical parameters such as Total Cholesterol, Triglycerides, LDL-C, HDL-C, VLDL-C, Serum Amylase, Serum Lipase and WBC Count were estimated to appraise its activity. A significant increase in the levels of biochemical and biological parameters was discerned in group 2 and a reversal in the extent was seen in the treated group of animals engaged in the study. The histopathological evaluations of Liver and Pancreas samples were in support of the obtained results. Based on the above results it can be suggested that *Lasia spinosa* leaves possess anti Hyperlipidemic activity and has the potential in prevention of hyperlipidemia induced pancreatitis.

INTRODUCTION: Herbal medicines and the use of medicinal herbs in the prevention and treatment of various ailments has been an archaic tradition in the history of human civilization. The progress being made and with increasing interests in the use of medicinal herbs among the health care professionals, the natural products are being used worldwide for diverse ailments and diseases. The annual global market of herbal medicines is over 60 billion USD [1].

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Hyperlipidemia means an increase in the levels of lipids in the body. The increased levels of cholesterol i.e. hypercholesterolemia and triglycerides i.e. hypertriglyceridemia in blood is responsible for various cardiovascular diseases and pancreatitis which has become a leading cause of death in many developed countries.

People with genetic susceptibility, dietary habits, stress, use of tobacco and alcohol are more prone to these diseases. Increased levels of lipids result in thrombus formation in the blood vessels leading to diseases of the coronary arteries which affects millions of people around the world [2]. Hence, increasing the awareness of hyperlipidemia in the public is an important factor in reducing its prevalence.

In human body, the role of the pancreas is to breakdown the carbohydrates, fats and proteins. When hyperlipidemia occurs, the lipids along with reactive oxygen species (ROS) causes the infection of pancreas known as pancreatitis which can be life threatening. An increase in the serum triglyceride levels to more than 1000 mg/dl is an identifiable risk factor for pancreatitis.[3] During pancreatitis abnormal levels of the pancreatic enzymes i.e. the serum amylase and serum lipase are present in the blood.

Lasia spinosa is a perennial tropical herb native to India, Southern china and Southeast asia.[4] It is commonly known as Kohila or Unicorn plant and belongs to the family Araceae.[5] It has been reported to contain Polyphenols, Ascorbic acid, Dietary fibers and Hydrocyanic acid.[6] Traditionally, the leaves of this herb are commonly employed in the treatment of gastrointestinal diseases, respiratory diseases and in skin infections.[6, 7] Since no evaluation of the Antihyperlipidemic activity of this plant being done before was found, the present study was carried out.

MATERIALS AND METHODS:

Collection and authentication of the plant:

Leaves of *Lasia spinosa* plant were obtained from the forest patches of Karbi, Anglong district in Assam, India. The plant was taxonomically identified and authenticated by the Botanical Survey of India, Rangareddy, Hyderabad, India.

Animals:

Studies were carried out using Swiss albino rats weighing 180-200 Gms of either sex. They were procured from the local market. The experimental protocol was approved by the Institutional Animal Ethics Committee constituted as per the CPCSEA guidelines with a registration number of (1330/AC/10/CPCSEA) [8].

Preparation of the plant extract:

The leaves of the plant were dried under shade and then coarsely powdered with a mechanical grinder. The powder was extracted with 95% methanol in a soxhlet extractor. The concentrated extract was evaporated to dryness and the extract obtained was weighed and labeled as LS-ME [9].

Preliminary Phytochemical Screening:

The solution of the methanolic extract was prepared using distilled water and subjected to preliminary phytochemical screening. Test for common phytochemicals were carried out by standard methods described in practical pharmacognosy by Kokate and Khandelwal[10, 11].

Determination of Acute toxicity (OECD guideline 423):

The acute toxicity for methanolic extract of leaves of *Lasia spinosa* (LS-ME) was determined in albino rats following OECD guideline 423, maintained under standard conditions [8].

Evaluation of Antihyperlipidemic activity:

TABLE 1: CHOLESTEROL INDUCED HYPERLIPIDEMIC MODEL.[12]

Group no.	Treatment and Label	Dose (b.w. p.o.)
Group 1	Normal control.	Fed with basal diet and normal saline
Group 2	Hyperlipidemic control (untreated group)	Cholesterol at a dose of 100 mg/kg for 30 days.
Group 3	Test dose 1	Cholesterol as in group 2 and LS-ME at a dose of 200 mg/kg in normal saline from day 15 to day 30.
Group 4	Test dose 2	Cholesterol as in group 2 and LS-ME at a dose of 400 mg/kg in normal saline from day 15 to day 30.
Group 5	Test dose 3	Cholesterol as in group 2 and LS-ME at a dose of 800 mg/kg in normal saline from day 15 to day 30.
Group 6	Positive control	Cholesterol as in group 2 and Atrovastatin at 10 mg/kg in normal saline from day 15 to day 30.

TABLE 2: TRITON-X 100 INDUCED HYPERLIPIDEMIC MODEL.[13]

Group no.	Treatment and Label	Dose
Group 1	Normal control.	Fed with basal diet and normal saline.
Group 2	Hyperlipidemic control (untreated group)	Triton-X 100 at a dose of 480 mg/kg b.w. i.p. for 7 days
Group 3	Test dose 1	Triton-X 100 at a dose of 480 mg/kg b.w. i.p. and LS-ME at a dose of 200 mg/kg b.w. p.o. in normal saline from day 3 to day 7.

Group 4	Test dose 2	Triton-X 100 at a dose of 480 mg/kg b.w. i.p. and LS-ME at a dose of 400 mg/kg b.w. p.o. in normal saline from day 3 to day 7.
Group 5	Test dose 3	Triton-X 100 at a dose of 480 mg/kg b.w. i.p. and LS-ME at a dose of 800 mg/kg b.w. p.o. in normal saline from day 3 to day 7.
Group 6	Positive control	Triton-X 100 at a dose of 480 mg/kg b.w. i.p. and Atrovastatin 10 mg/kg b.w. p.o. in normal saline from day 3 to day 7.

The Total cholesterol, Triglycerides, HDL-C were calculated by using cholesterol estimation kits and the LDL-C and VLDL-C were calculated using Friedwald's formula. The serum amylase concentration was estimated by means of Phadeba's amylase test, the serum lipase concentration was estimated by colorimetric lipase test kit and the WBC levels were estimated by means of automated cell count chambers.

Statistical Analysis:

Results were expressed as Mean ± SEM. Statistical analysis were performed with Graph pad prism software using one way Analysis of Variance followed by Dunnett's *t*-test.

P values were considered significant when *P<0.05, **P<0.01, ***P<0.001 when the test and standard were compared with the untreated groups [8].

RESULTS AND DISCUSSION:

Preliminary Phytochemical Analysis:

Preliminary phytochemical screening of 95% Methanolic extract of *Lasia spinosa* extract showed the presence of Carbohydrates, glycosides, alkaloids, tannins, flavonoids and proteins.

Acute toxicity studies:

The acute toxicity studies of 95% methanolic leaves extract of *Lasia spinosa* was carried out as per OECD guideline no. 423. There was no gross evidence of any abnormality observed up to a period of 4-6 hrs or mortality up to a period of 24hrs at the maximum tolerated dose level of 2000 mg/kg body weight p.o. Further pharmacological screening were carried out with three dose ranges i.e. 200 mg/kg b.w. p.o., 400 mg/kg b.w. p.o. and 800 mg/kg b.w. p.o.

Effect of LS-ME in Cholesterol induced Hyperlipidemia in rats:

Biochemical parameters:

Serum Lipid Profile:

Cholesterol administration (100 mg/kg b.w. p.o.) to the rats resulted in impairment in normal lipid profile (Table 3) leading to increased Total cholesterol, Triglycerides, LDL-C and VLDL-C while HDL-C was decreased.

Total Cholesterol: A reversal ($p<0.05$) in the levels of total cholesterol was seen in the rats treated with LS-ME at 200 mg/kg. A significant reversal ($p<0.01$) was observed at 400 mg/kg (group 4) and 800 mg/kg b.w. p.o.(group 5). Highly significant reversal ($p<0.001$) was seen in the positive control group (Table 3), (group 6).

Triglycerides: A significant reversal ($p<0.01$) in the triglyceride levels, was observed in rats treated with LS-ME at a dose of 200 mg/kg (group 3), 400 mg/kg (group 4) and 800 mg/kg (group 5). Highly significant reversal ($p<0.001$) in triglyceride levels was observed in rats treated with the standard drug Atrovastatin at a dose of 10 mg/kg (group 6) body weight (Table 3).

HDL-C: No significant increase in the levels of HDL-C was found in the rats treated with the LS-ME at a dose of 200 mg/kg (group 3). A significant reversal ($p<0.01$) was seen at 400 mg/kg (group 4), 800 mg/kg b.w. P.o (group 5) and in the positive control group (**Table 3**), (group 6).

LDL-C: A significant reversal ($p<0.05$) in the levels of LDL-C was seen in the animals treated with LS-ME at 200 mg/kg b.w. p.o. (Table 3), (group 3). A significant reversal ($p<0.01$) in the levels of LDL-C was observed at 400 mg/kg (group 4) b.w. p.o. and 800 mg/kg (group 5). A highly significant reversal ($p<0.001$) in the levels of LDL-C was seen in the positive control group (group 6).

VLDL-C: A significant reversal ($p<0.05$) in the levels of VLDL-C levels was observed in the animals treated with LS-ME at 200 mg/kg (group 3) b.w. p.o. A significant reversal ($p<0.01$) was

seen at 400 mg/kg b.w. p.o. (group 4), 800 mg/kg (group 5) b.w. p.o. and in the positive control group (**Table 3**), (group 6).

TABLE 3: EFFECT OF LS-ME ON TOTAL CHOLESTEROL, TRIGLYCERIDES, HDL-C, LDL-C AND VLDL-C LEVELS OF RATS IN CHOLESTEROL INDUCED HYPERLIPIDEMIC MODEL

Group	Total cholesterol (mg/dl) Mean±SEM	Triglycerides (mg/dl) Mean±SEM	HDL-C (mg/dl) Mean±SEM	LDL-C (mg/dl) Mean±SEM	VLDL-C (mg/dl) Mean±SEM
Group 1 (normal control)	88.27±0.25	123.05±0.4	42.15±2.3	21.09±0.29	25.03±1.3
Group 2 (negative control)	248.61±0.6[a]	257.54±0.36[a]	26.38±0.17[a]	170.68±0.66[a]	51.55±0.35[a]
Group 3 (LS-ME 200mg/kg)	204±1.9*	214.15±0.83**	28.97±0.23[ns]	132.43±0.49*	42.71±0.26*
Group 4 (LS-ME 400mg/kg)	190.06±1.6**	168.12±2.19**	30.42±2.1**	126.54±0.23**	33.12±0.25**
Group 5 (LS-ME 800mg/kg)	136.41±2.6**	139.38±0.45**	34.63±0.36**	74.06±0.49**	27.72±0.9**
Group 6 (Positive control)	93.30±0.5***	128.31±0.71***	40.92±0.27**	26.97±0.25***	25.41±0.22**

All the values are Mean ± SEM., N=6; a compared to normal group ($p<0.001$), significances values are ***$p<0.001$, **$p<0.01$, *$p<0.05$ (versus Negative control group)

Effect of LS-ME in Triton-X 100 induced Hyperlipidemia in rats:

Biochemical Parameters:

Serum Lipid Profile:

Triton-X administration to the rats (480 mg/kg b.w. i.p) resulted in impairment in normal lipid profile leading to a highly increase in the levels of triglycerides, LDL-C and VLDL-C while HDL-C was decreased (**Table 4**).

Total Cholesterol: No significant reversal in the levels of total cholesterol was observed in the rats treated with LS-ME at 200 mg/kg (**Table 4**), (group 3). A significant reversal ($p<0.01$) was observed at 400 mg/kg (group 4) and 800 mg/kg (group 5) b.w. p.o. Highly significant reversal ($p<0.001$) was seen in the positive control group (group 6).

TABLE 4: EFFECT OF LS-ME ON TOTAL CHOLESTEROL, TRIGLYCERIDES, HDL-C, LDL-C AND VLDL-C LEVELS OF RATS IN TRITON-X 100 INDUCED HYPERLIPIDEMIC MODEL

Group (n=6)	Total Cholesterol (mg/dl) Mean±SEM	Triglycerides (mg/dl) Mean±SEM	HDL-C (mg/dl) Mean±SEM	LDL-C (mg/dl) Mean±SEM	VLDL-C (mg/dl) Mean±SEM
Group 1 (normal control)	88.27±0.25	123.05±0.4	42.15±2.3	21.09±0.29	25.03±1.3
Group 2 (negative control)	1018.12±1.69[a]	1287.97±18.95[a]	19.61±0.44[a]	729.65±3.09[a]	268.86±1.54[a]
Group 3 (LS-ME 200mg/kg)	910.12±2.69[ns]	1016.91±5.54*	20.05±0.42[ns]	689.86±1.03[ns]	200.21±1.24*
Group 4 (LS-ME 400mg/kg)	641.11±0.79**	872.94±2.80**	22.81±0.58*	474.14±1.51**	144.16±1.29**
Group 5 (LS-ME 800mg/kg)	495.10±1.28**	714.99±4.12**	25.17±1.45**	362.62±1.23**	107.31±1.17**
Group 6 (Positive control)	391.58±1.09***	406.67±2.18***	27.94±0.63**	268.23±1.35***	95.41±1.29***

All the values are Mean ± SEM., N=6; a compared to normal group ($p<0.001$), significances values are ***$p<0.001$, **$p<0.01$, *$p<0.05$ (versus Negative control group)

Triglycerides: A significant reversal ($p<0.05$) in the levels of triglycerides was observed in rats treated with LS-ME at 200 mg/kg b.w. p.o. (Table 4), (group 3). A significant reversal ($p<0.01$) was observed at 400 mg/kg (group 4) and 800 mg/kg b.w. p.o. (group 5). Highly significant reversal ($p<0.001$) in the triglyceride levels was seen in the positive control group (group 6).

HDL-C: No significant reversal in the levels of HDL-C was observed in rats treated with LS-ME at 200 mg/kg b.w. p.o.(group 3). A significant reversal ($p<0.05$) was observed at 400 mg/kg b.w. p.o (Table 4), (group 4). A significant reversal ($p<0.01$) in HDL-C was seen in rats treated with LS-ME at 800 mg/kg b.w. p.o.(group 5) and in positive control group (group 6).

LDL-C: No significant reversal in the levels of LDL-C was observed in rats treated with LS-ME at 200 mg/kg b.w, p.o.(group 3). A significant reversal ($p<0.01$) was observed at 400 mg/kg (group 4) and 800 mg/kg b.w. p.o (Table 4), (group 5). Highly significant reversal ($p<0.001$) was seen in positive control group (group 6).

VLDL-C: A significant reversal ($p<0.05$) in the levels of VLDL-C was observed in rats treated with LS-ME at 200 mg/kg b.w. p.o.(group 3). A significant reversal ($p<0.01$) was observed at 400 mg/kg b.w. p.o. (group 4) and 800 mg/kg (group 5) b.w. p.o. A significant reversal ($p<0.001$) was seen in positive control group (group 6).

Determination of serum amylase Concentration: The rats treated with LS-ME at a dose of 200 mg/kg (**Table 5**), (group 3) and 400 mg/kg b.w, p.o.(group 4) showed a significant reversal ($p<0.05$) in serum amylase concentration. With LS-ME at 800 mg/kg (group 5), there was a significant reversal ($p<0.01$) in the serum amylase concentrations. A highly significant reversal ($p<0.001$) in the serum amylase concentration was observed in the rats treated with the standard drug Atrovastatin at a dose of 10 mg/kg (group 6)

TABLE 5: EFFECT OF LS-ME ON SERUM AMYLASE LEVELS OF RATS IN TRITON-X 100 INDUCED HYPERLIPIDEMIC MODEL

Group (n=6)	Serum Amylase concentration(U/L) Mean±SEM
Group 1 (Normal control)	18.83±1.09
Group 2 (Negative control)	461.67±16.10[a]
Group 3 (LS-ME 200mg/kg)	416.34±12.82*
Group 4 (LS-ME 400mg/kg)	373.48±8.57*
Group 5 (LS-ME 800mg/kg)	206.59±10.82**
Group 6 (Positive control)	152.81±1.49***

All the values are Mean ± SEM., N=6; a compared to normal group ($p<0.001$), significances values are ***$p<0.001$, **$p<0.01$, *$p<0.05$ (versus Negative control group)

Determination of Serum Lipase Concentration: The rats treated with LS-ME at a dose of 200 mg/kg (**Table 6**), (group 3) body weight p.o. showed a significant reversal ($p<0.05$) in the Serum lipase concentrations. A significant reversal ($p<0.01$) in the serum lipase concentrations was observed in the group of rats treated with LS-ME at 400 mg/kg b.w. p.o. (group 4) and 800 mg/kg (group 5) b.w. p.o.

Highly significant reversal ($p<0.001$) in the serum lipase concentrations was observed in the rats treated with the standard drug Atrovastatin (group 6) at a dose of 10 mg/kg b.w. p.o.

TABLE 6: EFFECT OF LS-ME ON SERUM LIPASE LEVELS OF RATS OF TRITON-X 100 INDUCED HYPERLIPIDEMIC MODEL

Group (n=6)	Serum Lipase concentration (U/L) Mean±SEM
Group 1 (Normal control)	31.79±4.63
Group 2 (Negative control)	231.19±29.72[a]
Group 3 (LS-ME 200mg/kg)	187.63±21.86*
Group 4 (LS-ME 400mg/kg)	119.32±14.91**
Group 5 (LS-ME 800mg/kg)	86.27±9.31**
Group 6 (Positive control)	71.57±4.46***

All the values are Mean ± SEM., N=6; a compared to normal group ($p<0.001$), significances values are ***$p<0.001$, **$p<0.01$, *$p<0.05$ (versus Negative control group)

Determination of Differential WBC Count:

A significant reversal ($p<0.05$) in the no. of white blood cells was seen in the rats treated with LS-ME at a dose of 200 mg/kg b.w. p.o. (**Table 7**), (group 3). A significant reversal ($p<0.01$) in the no. of WBC was seen in the rats treated at 400 mg/kg (group 4) b.w. p.o. and 800 mg/kg b.w. p.o. Highly significant reversal ($p<0.001$) in the no. of WBC was seen in the positive control group (group 6) b.w. p.o.

TABLE 7: EFFECT OF LS-ME ON WBC LEVEL OF RATS IN TRITON-X 100 INDUCED HYPERLIPIDEMIC MODEL

Group (n=6)	Differential White Blood cell Count (cells/mcl)
Group 1 (Normal control)	7,500±100
Group 2 (Negative control)	42,800±200[a]
Group 3 (LS-ME 200mg/kg)	34,100±900*
Group 4 (LS-ME 400mg/kg)	21,900±400**
Group 5 (LS-ME 800mg/kg)	16,700±500**
Group 6 (Positive control)	10,700±300***

All the values are Mean ± SEM., N=6; a compared to normal group ($p<0.001$), significances values are ***$p<0.001$, **$p<0.01$, *$p<0.05$ (versus Negative control group).

Histopathology:

Hepatic Histopathology:

On external observation, the livers of the untreated group (group 2) were found to be more bulky whereas the livers of the treated groups were found to be less bulky when compared to the untreated livers in Cholesterol induced hyperlipidemic model (**Figure 1).**

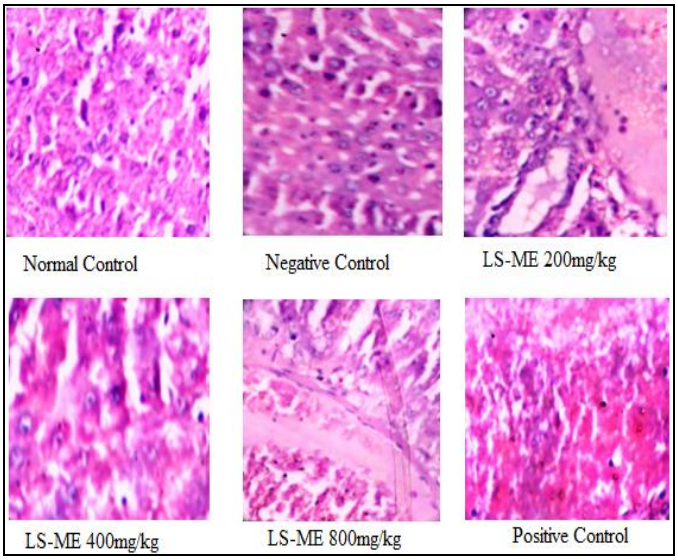

FIG.1: HISTOPATHOLOGY OF LIVER SAMPLES (10X) IN CHOLESTEROL INDUCED HYPERLIPIDEMIC MODEL

The livers of untreated rats exhibited a typical sign of fatty liver showing the accumulation of fat droplets through the liver acini was found in case of Triton-X 100 (**Figure 2**) induced hyperlipidemic model. When rats were treated with LS-ME or Atrovastatin, however a smaller degree of lipid accumulation and fewer pathological signs were observed in a dose dependant manner.

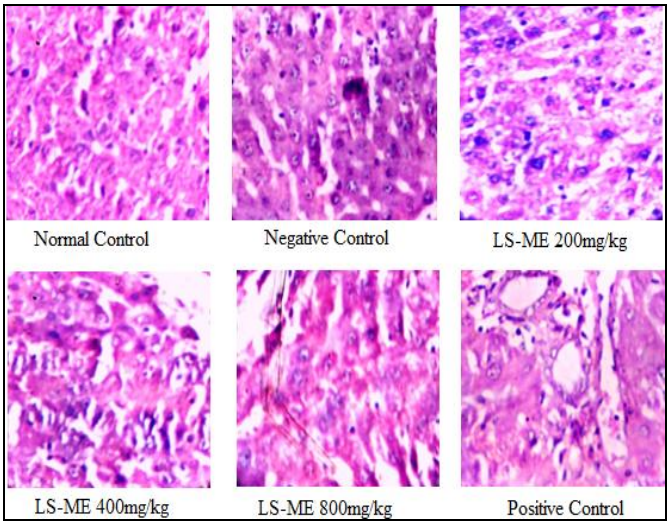

FIGURE 2: HISTOPATHOLOGY OF LIVER SAMPLES (10X) IN TRITON-X 100 INDUCED HYPERLIPIDEMIC MODEL

Pancreatic Histopathology:

A prominent thickening of the wall of the pancreatic artery with infiltration by mononuclear cells and peri-arterial fibrosis was found in the untreated group. Parenchymal damage was also seen. Whereas the pancreatic treated groups showed less parenchymal damage in a dose dependant manner (**Figure 3**). The anti-hyperlipidemic activity of 95% methanolic leaves extract of *Lasia spinosa* was evaluated by employing Cholesterol induced hyperlipidemic model and Triton-X 100 hyperlipidemic model. Triton-X 100 induces acute hyperlipidemia by raising the serum lipoproteins levels. It acts on serum lipoprotein lipase thereby inducing hyperlipidemia.

The data obtained in the present study revealed that there was a significant increase in the concentration of lipids in the untreated group (group 2) of animals. The biochemical parameters were decreased in the group of rats treated with the plant extract of *Lasia spinosa* (groups 3-5) in a dose dependant manner.

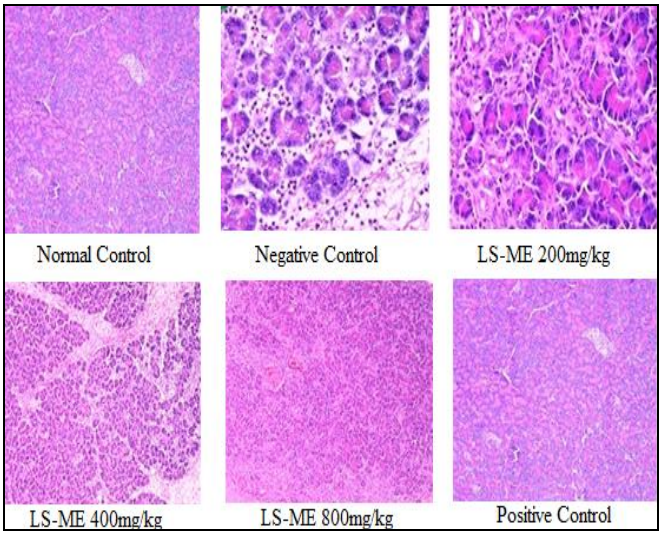

FIGURE 3: HISTOPATHOLOGY OF PANCREAS (10X) IN TRITON-X 100 INDUCED HYPERLIPIDEMIC MODEL

CONCLUSION: The Phytochemical screening results indicated the presence of alkaloids, glycosides, saponins, tannins, carbohydrates proteins and flavonoids. Ascorbic acid is considered as a well known antioxidant. The acute toxicity studies for LS-ME were carried out and no gross evidence of any abnormalities or mortality were found in the rats even at a maximum tolerated dose levels of 2000 mg/kg body weight p.o.

In conclusion, the finding in this study suggests that the *Lasia spinosa* leaves possess anti hyperlipidemic activity and has the potential in prevention of hyperlipidemia induced pancreatitis in rats. Considering the significant increase in serum HDL cholesterol in the animals treated with LS-ME, which is a cardio protective lipid, studies, can be carried out on the LS-ME extract to be concluded as a cardio protective agent apart from being an anticestodal and Antihyperlipidemic agent. Further, studies can be extended to determine the exact mechanism of action for its anti-hyperlipidemic activity.

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