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HYPOLIPIDEMIC EFFECT OF HYDRO-ETHANOLIC EXTRACT OF AMORPHOPHALLUS CAMPANULATUS IN ETHANOL INDUCED DYSLIPIDEMIA IN MALE WISTAR RAT

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ABSTRACT Chronic alcohol consumption causes alcoholic liver disease, which is associated, or initiated, with dysregulated lipid metabolism. Hyperlipidemia is a lipid metabolism disorder and the major risk factor for the development of CVDs. The current study is designed to emphasize the hypolipidemic effect of hydro-ethanolic extract of Amorphophallus campanulatus (AC) in ethanol administered male Wistar rat model. Male Wistar rats weighing 150-200gm were used in the study and were divided into control, ethanol treated, extract treated (250 and 500mg/kg body weight) and atorvastatin (10mg/kg body weight) treated groups. The ethanol treated groups were administered 40% ethanol 2gm/kg body weight while the extract and standard drug treated groups were simultaneously treated with the similar dose of ethanol prior to extract and drug treatment. The normal controls received equivalent amount of normal saline. Long-term excessive alcohol feeding to rats caused fatty liver and liver injury, which was associated with disrupted cholesterol homeostasis, characterized by increased hepatic cholesterol levels and hypercholesterolemia. Ethanol administration elevates total cholesterol by 54.96%, triglyceride and VLDL by 46.67% and LDL by 79.53% as compared to control group. However, in contrast to these the HDL cholesterol is significantly lowered ($P<0.001$) in the ethanol treated group as compared to control by 46.82%. Administration of hydro-ethanolic extract of AC (250 mg/kg and 500 mg/kg) together with continuous ethanol administration for four weeks showed significant reduction in serum cholesterol level by 25.5% and 37.1%, respectively ($p < 0.05$); serum triglyceride level by 22.6% and 33.6%, respectively ($p < 0.05$); serum LDL level by 24.1% and 35.4%, respectively ($p < 0.05$), and serum VLDL level by 15.5% and 20.5%, respectively ($p < 0.05$), as compared to ethanol group. However significant change ($p < 0.05$) in HDL level was also observed. AC 500 mg/kg was more effective than AC 250 mg/kg. AC 500mg/kg dose was as effective as the standard drug, atorvastatin. Histological study also showed that ethanol group had fatty infiltration in liver sections which was arrested in the extract treated groups. Thus the results of the study indicate that Amorphophallus campanulatus extract show considerable hypolipidemic action that make it encouraging as future therapeutic agent to treat dyslipidemia.

KEYWORDS : Alcohol, Dyslipidemia, Ethanolic Extract, Hypercholesterolemia

INTRODUCTION

Dyslipidemia or hyperlipidemia can result from an intrinsic, extrinsic, or a combination of genetic predisposition and external factors. Initial dyslipidemias are heterogeneous diseases with genetic, monogenic, or polygenic etiologies, while secondary dyslipidemias result from the association of risk factors with external factors. Among many such external factors, chronic ethanol intake is well known to cause deregulated lipid metabolism. In addition to disturbing triglyceride metabolism, chronic consumption of excessive alcohol can also exert adverse effects on cholesterol metabolism. Besides, excessive triglyceride accumulation in hepatocytes due to alcohol abuse not only is the initial stage of disease spectrum of alcoholic liver disease, but also plays a critical role in the disease progression to late stage hepatitis and/or cirrhosis. It has been reported previously that ethanol feeding increased hepatic esterified cholesterol levels in rats, and this was associated with both enhanced cholesterol biosynthesis and decreased bile acid excretion (1). Hyperlipidemia characterized by elevation of cholesterol, cholesterol esters, phospholipids, or triglycerides is an additional outcome of alcohol abuse. Apart from the burden of alcoholic liver disease (ALD), abnormalities of plasma lipids can result in predisposition to coronary, cerebrovascular, and peripheral vascular arterial diseases (2). Lipids and lipoprotein abnormalities are preceding risk factor for cardiovascular diseases and prevalence of this in general population has increased considerably in last few decades. Hyperlipidemia contributes significantly in the prevalence and severity of atherosclerosis and coronary heart diseases (3). Cardiovascular diseases are the primary cause of mortality and morbidity worldwide (4). Case-control and cohort studies of middle-aged populations have consistently demonstrated relationships between alcohol consumption and the incidence of major vascular diseases in particular coronary heart disease.

Multiple factors trigger the onset of such vascular diseases, of which there exists strong contribution of endothelial damage related to lipid peroxidation. This endothelial dysfunction increases the permeation of low-density lipoproteins (LDL) through the intima layer, resulting in oxidation and formation of atherosclerotic damage (5). In order to control this imbalance, the body has enzymatic and nonenzymatic antioxidant defense mechanisms capable of preventing the deleterious effects of oxidation, inhibiting lipid peroxidation, free radicals scavenging, and maintaining redox balance in cells.

In addition to endogenous antioxidants, there are antioxidants from exogenous sources. The beneficial effects of foods have been linked to the presence of bioactive compounds and other nutrients. Amorphophallus campanulatus, a rhizomatous or tuberous perennial from the family Araceae has its mention in Ayurveda. Traditionally the root is dried and used in the treatment of piles and dysentery. The fresh root acts as an acrid stimulant and expectorant, it is much used in India in the treatment of acute rheumatism. Especially in Ayurveda, it is used in arthralgia, elephantiasis, tumors, inflammations, hemorrhoids, hemorrhages, vomiting, cough, bronchitis, asthma, dyspepsia, colic, constipation, hepato-splenopathies, amenorrhea, dysmenorrhea, seminal weakness, fatigue and general debility. This is also a starch based food item but it has several important antinutrient components like steroids, alkaloids, tannins, glycosides, phenols, flavonoids, saponins etc. In previous studies conducted in our laboratory, the hydro-ethanolic extracts of this tuber showed in vitro and in vivo antioxidative properties as well as hepatoprotective properties against ethanol induced hepatotoxicity (6, 7).

In this context, the aim of this study was to evaluate the hypolipidemic activity of the hydro-ethanolic extract of Amorphophallus campanulatus in ethanol induced hyperlipidemic albino Wistar rats.

MATERIALS AND METHODS

Procurement of Plant Material

Fresh tubers of Amorphophallus campanulatus (AC) were purchased from the farmers of Santragachhi village of Howrah district, West Bengal, India, from the month of May to October. Authentication was done by the Central National Herbarium of Botanical Survey of India (BSI), Howrah, West Bengal, India. Herbarium of the specimen is maintained in the institute library bearing the number RMC/PHY/SB/03/14.

Preparation of Hydro-ethanolic Extract of Amorphophallus Campanulatus Tuber

Raw fresh tubers after procurement were cut into thin slices and washed under running tap water to remove any impurity and mud. After proper cleaning the wet materials were soaked in blotting paper and then dried in hot air oven at 50°Celsius, until materials became crispy in texture. These dried materials were grinded into coarse powder form and stored at -4° Celsius in air tight Tarson jars until further use.

For the preparation of the hydro-ethanolic extract, 20 gm of the powdered dried samples of AC were taken in the thimble of Soxhlet and extracted with 250 ml of ethanol (70%) in the round bottomed flask continuously for a week. The extracts were then filtered through muslin cloth, centrifuged and the collected filtrates were first concentrated followed by evaporation to dryness using rotary evaporator (M/s B.C. Chatterjee & Co., Kolkata, West Bengal, India) at 50° Celsius. The dried samples, hydro-ethanolic extracts of and Amorphophallus campanulatus (HEEAC) were collected and stored in air tight plastic vials at -4° Celsius for future use. The dried sample was dissolved in normal mammalian saline (0.9 gm% NaCl) at concentrations of 250 and 500 mg/kg body wt for administration to experimental animal groups.

Animal Selection, Care and Maintenance

Wistar strain male Albino rats weighing 170-200 g body weight were procured from authorized breeders (Kolkata, India). The rats were housed in autoclavable polypropylene cages and maintained under temperature controlled room (25˚±2˚C) with 12:12 hour L:D photoperiod. Rats were given standard pellet diet (Lipton, Rat Feed, Ltd., Pune, India) and water ad libitum throughout the experimental period. All experiments were in accordance with the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animal (CPCSEA), Government of India [1795/PO/ERe/S14CPCSEA].

Toxicity Determination and Dose Standardization of Ethanolic Extract of Amorphophallus Campanulatus Tuber

For inducing hepatotoxicity the dose of ethanol was determined to be 40% v/v ethanol, at 2 g per kg body weight (6). As described in the guidelines of OECD (TG 407, OECD, 2008) given by CPCSEA, groups of rats (n = 6) were administered the extract (i.p.) at doses ranging from 1000 to 6000 mg/kg body weight, and their mortality was detected. The calculated LD50 value, according to Lorke's method, (1983) (8, 9) was found to be 5477.22 mg/kg. The HEEAC was reflected as non-toxic. For further study, we selected 1/20th and 1/10th of the maximum safe dose (5,000 mg/kg).

Experimental Design

The animals were divided into the following groups:-

Group I (Cont): Standard diet + equal volume of normal saline (0.9gm% NaCl).

Group II (Eth): Standard diet + Standardized doses of ethanol (2gm/kg body weight).

Group III (Eth+HEEAC250): Standard diet + Standardized doses of ethanol (2gm/kg body weight) + Standardized dose of HEEAC (250 mg/kg body weight)

Group IV (Eth+HEEAC500): Standard diet + Standardized doses of ethanol (2gm/kg body weight) + Standardized dose of HEEAC (500 mg/kg body weight)

Group V (HEEAC250): Standard diet + Standardized dose of HEEAC (250 mg/kg body weight)

Group VI (HEEAC500): Standard diet + Standardized dose of HEEAC (500 mg/kg body weight)

Group VII (Eth+Ator10): Standard diet + Standardized doses of ethanol (2gm/kg body weight) + Atorvastatin 10mg/kg body weight)

Collection of Sample

After thirty days of continuous treatment, the animals were kept fasted overnight without any treatment. The day following, the animals were sacrificed after being anaesthetized with an intraperitoneal injection of a combination of 100mg/kg of ketamine and 10mg/kg Xyalazine (10). Blood samples were collected by cardiac puncture. The collected blood was allowed to stand for some time to separate out the serum. Clear serum was obtained after centrifugation of collected blood specimens at 3500 rpm for 20 minutes using mini centrifuge (M/s Remi Laboratory Instruments, Model R-303). Liver from different experimental groups were excised, trimmed of with filter paper and weighed to obtain the organ weight. Part of the excised livers was used for homogenate preparation and the other part was transferred to fixative for preparation of histological sections.

Measurement of Liver Weight to Body Weight Ratio

Body weight of all animals at the end of the treatment period, along with their respective liver weight on the day of sacrifice was noted to determine the liver weight to body weight ratio of animals in each group.

Assessment of Antioxidant Status

Preparation of Tissue Homogenate

A 10% w/v liver tissue homogenate was prepared in ice cold Phosphate buffer saline (PBS) containing 1mM EDTA (pH 7.4), using tissue homogenizer. The homogenate was then centrifuged at 10,000 rpm for 30 min, at 4 °C. The supernatant thus obtained was used further, for the following biochemical assay of tissue oxidative stress markers.

Estimation of Lipid Peroxidation (LPO)

Lipid peroxidation was estimated by measuring thiobarbituric acid reactive substance (TBARS), according to the method of Buege and Aust, 1978 (11). 1 ml of cytosol was mixed with 10% TCA and 0.67% TBA reagent, and boiled for 20 min in water bath. The samples were then cooled and centrifuged at 3000 rpm for 10 min at room temperature. The optical density of the pink chromogen present in the clear supernatant was measured at 532 nm using a UV–VIS spectrophotometer. The values were calculated using molar extinction co-efficient, and expressed as µmoles of malondialdehyde (MDA)/100 gm tissue.

Estimation of Cytosolic Superoxide Dismutase (Cu-Zn SOD)

Cu-Zn SOD activity was measured according to the method of Marklund and Marklund 1974 (12). 2 ml of Tris-buffer containing 0.1mM EDTA was mixed 10 µL of tissue cytosol and the reaction was initiated by adding pyragallol (20mM in Tris-HCl buffer, pH 6.6). The absorbance was then measured at 420 nm for 5 min, at an interval of every1 min. The enzymatic activity was expressed as U/min/mg protein of liver extract and 1 U of enzyme is defined as the enzyme activity that inhibits auto-oxidation of pyragallol by 50%.

Estimation of Catalase (CAT)

Catalase activity was determined by the decomposition of H_2O_2 at 240 nm, according to the method of Beers and Sizer 1952 (13). 3 ml of H_2O_2 solution (10 mmol/L in 50 mmol/L potassium phosphate buffer, pH 7.0) was mixed with 1 µL of cytosol and the decrease of absorbance in every 30 s was recorded over a period of 3 min. Change in the rate of absorbance was converted µmoles of H2O2/min/mg protein.

Estimation of Reduced Glutathione (GSH)

Reduced glutathione content was estimated according to the method of Sedlak and Lindsay 1968 (14). 1 ml cytosol was deproteinized using 10% TCA. It was then centrifuged and the supernatant obtained was used for the assay. 0.5 ml of the clear supernatant was diluted with phosphate buffer containing 1mM EDTA (pH 8), and then DTNB was added for colour development. After incubation for 30 min the absorbance was measured at 412 nm and the values were calculated using molar extinction co-efficient, and expressed as mg/100 gm tissue.

Estimation of Glutathione Peroxidase (GPx)

Glutathione Peroxidase activity was measured according to the method of Rotruck et al. 1973 (15). 200 µL of cytosol was diluted with 0.4M phosphate buffer (pH 7.0) and mixed with 100 µL of Sodium azide (10 mM), 200 µL of Glutathione (4 mM) and finally, 100 µL of (0.2 mM) H2O2 was added into it and incubated at 37 °C for 10 min. The reaction was then stopped by adding 400 µL of 10% TCA. It was then centrifuged at 3000 rpm for 20 min, and the clear supernatant obtained was assayed for GSH content using DTNB. Glutathione Peroxidase activity was expressed as µmoles of GSH consumed/min/mg protein.

Estimation of Glutathione Reductase (GR) Activity

Glutathione Reductase was measured by the method of Racker (16). 3 ml reaction mixture contains 1.7ml 0.01M sodium phosphate buffer (pH 7.0), 400µl of 1mM EDTA, 400µl of 1mM GSSG and 400µl of 2 mM NADPH. The reaction was started with the addition of 0.1ml of tissue supernatant (PMS). The decrease in OD/ min was noted and followed at every l min interval for 5 minutes at 340 nm.

Assessment of Lipid Profile

Lipid profiling was done by using commercially available kits. Total Cholesterol (CHLS), was estimated by cholesterol oxidase-peroxidase (CHOD-PAP) enzymatic end point assay method (16). Triglyceride (TG), was estimated by glycerol phosphate oxidase (GPOPAP) method (17). Then the High density lipoprotein (HDL), Low density lipoprotein (LDL) and Very low density lipoprotein (VLDL) contents were calculated from CHLS and TG levels, using equations mentioned in manufacturer's manual.

Estimation of Serum and Tissue Protein

Tissue protein was estimated according to the method of Lowry et al. 1951 (18) using BSA as standard. All absorbance at different

wavelengths were carried out using UV–VIS spectrophotometer (Systronics 118).

Histopathalogical Studies

Liver of sacrificed rats was carefully dissected out. After rinsing in normal saline the tissue was fixed in 10% formalin-saline dehydrated with 100% ethanol solution and embedded in paraffin. Then it was cut into 4-5µ thick sections and stained with hematoxylin-eosin and observed under microscope.

RESULTS

Effect of HEEAC on Liver Weight to Body Weight Ratio

Table 1 represents the liver weight to body weight ratio of the experimental animal groups. It is observed that the liver weight as well as the ratio of liver weight to body weight significantly ($p<0.001$) increased in ethanol (2gm/kg body weight) treated group. Consequently, co-administration of ethanolic extracts of HEEAC (250 and 500 mg/kg body weight), and atorvastatin (10mg/kg body weight) along with ethanol showed significant decrease ($p<0.01$) in liver weight and ratio of liver weight to body weight as compared to the ethanol treated group. Liver weight and ratio of liver weight to body weight shows no significant variation among the only extract and atorvastatin treated groups when compared to control. The altered liver weight to body weight ratio due to ethanol is restored near normal when supplemented with both doses of HEEAC (250mg and 500mg/kg body weight) when compared to control.

Table 1: Effect of HEEAC on Liver Weight and Liver wt to Body wt Ratio in the Control and Experimental Rats.

Groups	Initial Body Weight (gm)	Final Body Weight (gm)	Liver Weight (gm)	Liver Wt to Body Wt Ratio (%)
Group I (Cont)	150.83 ± 4.12	175.64 ± 5.02	5.6 ± 1.15	3.20 ± 0.18
Group II (Eth)	155.25 ± 5.92	165.49 ± 6.19	6.9 ± 1.58*	4.18 ± 0.11*
Group III (Eth+HEEAC250)	160.22 ± 5.66	170.44 ± 4.16	5.5 ± 2.14**	3.29 ± 0.06**
Group IV (Eth+HEEAC500)	158.34 ± 6.08	168.84 ± 6.08	5.6 ± 1.24** ns	3.21 ± 0.17** ns
Group V (HEEAC250)	156.29 ± 4.62	170.24 ± 5.22	5.8 ± 1.18**ns	3.35 ± 0.13**ns
Group VI (HEEAC500)	153.66 ± 5.25	165.00 ± 7.42	6.38 ± 1.90	3.25 ± 1.44**ns
Group VII (Eth+Ator10)	150.50 ± 4.89	160.80 ± 5.14	5.11 ± 1.64**ns	3.28 ± 1.38**ns

All values are expressed as MEAN ± SD, of 6 animals in each group. Data are analyzed by one way analysis of variance (ANOVA) followed by Tukey's Kramer post hock analysis. * P<0.001 vs. control. ns P>0.05 vs. control, **P<0.01 vs. only ethanol

Effect of Hydro-ethanolic Extract of (HEEAC) on the Lipid Profile in Control and Experimental Rats.

In this study it is shown in Figure 1 how ethanol administration alters the total lipid profile in experimental group as compared to control. It is observed that total cholesterol, LDL cholesterol, VLDL cholesterol and triglyceride all are significantly (P<0.001) elevated upon prolonged and continuous ethanol treatment. Ethanol administration elevates total cholesterol by 54.96%, triglyceride and VLDL by 46.67% and 35.59% respectively and LDL by 79.53% as compared to control group. These elevated lipid profile observed with ethanol treatment can be due to ethanol's' enhancing effect on lipid biosynthesis and an overall positive balance for lipid anabolism and negative balance for lipid catabolism. However, in contrast to these the HDL cholesterol is significantly lowered (P<0.001) in the ethanol treated group as compared to control by 46.82%. The study reveals that on administration of HEEAC at two different concentrations, such elevation in TC, TG, LDL and VLDL is significantly (P<0.001) restricted and prevented and at the same time lowering of HDL is also significantly (P<0.001) attenuated as compared to the ethanol administered group. Thus the extracts show potent hypolipidemic action against ethanol induced hyperlipidemia. The capacity of the extracts to prevent such alterations in lipid profile is comparable with that of the standard hepatoprotective drug atorvastatin. So proper functioning of hepatocytes is also manifested in maintaining ideal lipid profile by the extract treated animals. The only extract treated groups also show similar TC, TG, HDL, LDL and VLDL levels with non-significant difference (P>0.05) as compared to control. Another calculated parameter observed in this study is atherogenic co-efficient which is an indicator of predisposition to cardiovascular disorder. Higher atherogenic co-efficient is indicative of greater risk to cardiovascular disorders (19). The study focuses on the fact that ethanol administered animals have significantly (P<0.001) higher atherogenic co-efficient compared to control. This risk of ethanol to induce cardiovascular hazard is significantly lowered (P<0.001) when co-administered with the two different doses of HEEAC (250mg and 500mg/kg body weight). The only extract treated groups show no significant (P>0.05) difference in atherogenic co-efficient as compared to control.

All values are expressed as MEAN ± SD, of 6 animals in each group. Data are analyzed by one way analysis of variance (ANOVA) followed by Tukey's Kramer post hock analysis. * P<0.001 vs. control. ns P>0.05 vs. control, **P<0.01 vs. only ethanol

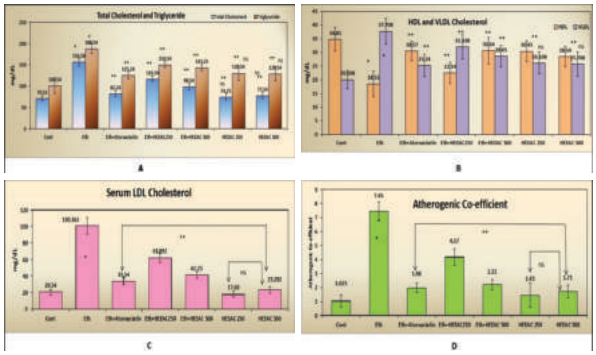


Figure 1: Changes in Lipid Profile and Impact on Atherogenic Index in Control and Experimental Animals.

Figure 2 represents the antioxidant milieu of the hepatocytes depicting the changes in the kinetics of critical enzymatic antioxidants like Cu/Zn SOD, catalase, GPx and GR as well as non-enzymatic antioxidant GSH. Another crucial indicator of the oxidative imbalance is the lipid peroxidation products (LPO) is also measured in the study. It is observed that levels of hepatic GSH, Cu/Zn SOD and catalase in the control group receiving mammalian normal saline is considered the basal level and upon administration of ethanol for thirty consecutive days the levels of these parameters significantly (P<0.001) decreases as compared to the basal.

All values are expressed as MEAN ± SD, of 6 animals in each group. Data are analyzed by one way analysis of variance (ANOVA) followed by Tukey's Kramer post hock analysis.
*P<0.001 vs. control. ns P>0.05 vs. control, **P<0.01 vs. only ethanol

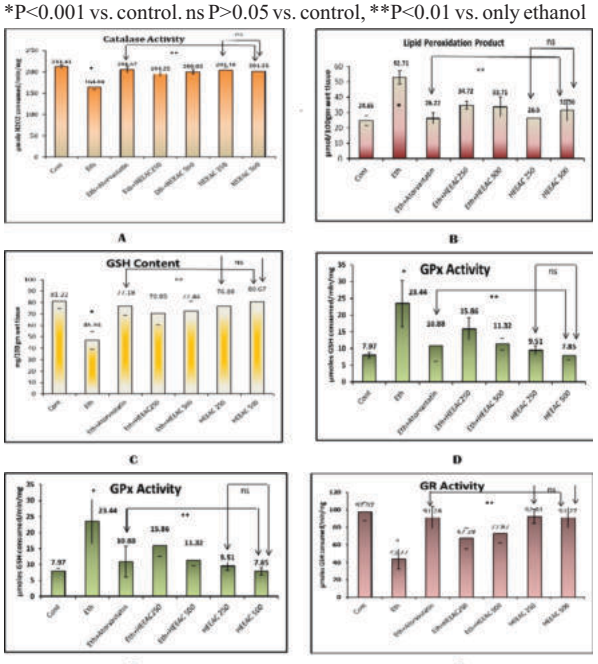
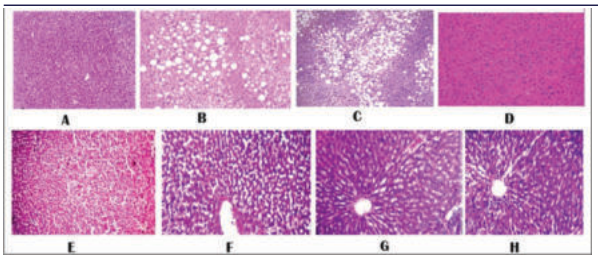


Figure 2: Haematoxylin-Eosin Staining of Hepatic Tissue Slices From Different Experimental Group of Animals at 100X



A: Control
B and C: Ethanol Treated, showing lipid droplet accumulation.
D: Ethanol along with Atorvastatin (10mg/kg body wt)
E: Ethanol along with HEEAC 250 mg/kg body wt
F: Ethanol along with HEEAC 500 mg/kg body wt
G: Only HEEAC 250 mg/kg body wt
H: Only HEEAC 500 mg/kg body wt

DISCUSSION

Alcohol intake is a public health concern and burden worldwide as it exerts detrimental effects on multiple organs possess serious injury to health including neural problems, reproductive issues, cardiovascular diseases, diabetes, suppression of the immune system etc. In the present study, we demonstrated that chronic alcohol exposure for 4 weeks resulted in disturbed cholesterol homeostasis in rats. In the present study it was observed that serum total cholesterol, triglyceride, LDL and VLDL were elevated in ethanol administered group. Exposure to alcohol has an impact on lipolysis, lipid profile and lipid peroxidation process and results in alcoholic hypercholesterolemia, hypertriglyceridemia. Circulating TGs have been positively correlated with acute and chronic ethanol toxicity in experimental animals and humans, possibly due to the decreased clearance of TGs from blood and/or decreased oxidation of fatty acids. Increased hepatic TGs block the TG synthesis leading to an increase in free fatty acids (FFAs). FFAs are known to contribute to fatty liver in ALD (20). The oxidation of alcohol to acetaldehyde concomitantly reduces NAD to NADH. The increased NADH promotes fatty acid synthesis and opposes lipid catabolism leading to fat accumulation in hepatocytes. In the current study the histological sections of liver tissue show fatty infiltration indicating early signs of fatty liver and steatohepatitis. The changes observed in our study are in correlation to the changes observed in ethanol consumers. Evidence suggests that fat accumulation in hepatic tissue is higher in ethanol consumers compared to that in non-drinkers (21).

Besides, fat accumulation sensitizes the liver to induce inflammation and cell death due to buildup of oxidative stress (22). Our study also provides evidence for oxidative stress in hepatic tissue by measuring the levels of lipid peroxidation products, reduced glutathione and enzymatic kinetics of SOD, catalase and GPx, the principle enzymatic antioxidant defense of the body. The study results reveal elevated levels of LPO, and increased enzymatic activity of SOD, catalase and GPx while a reduced level of GSH. An endogenous imbalance of free radicals result in oxidative stress which is manifested by lowered levels of GSH and enhanced activity of SOD, catalase and GPx, the free radical scavenging enzymes. Changes observed in cholesterol (free/esterified), fatty acids and TGs along with biochemical changes in antioxidant markers reveal an interplay that leads to ethanol induced steatosis followed by advanced progression of the disease towards fibrosis and cirrhosis. In our study, from the lipid profile values, we have also calculated the atherogenic index (AI), an indicator of the risk of developing cardiovascular disease (CVD). It is observed that the ethanol treated group had high AI compared to the other groups. Chronic ethanol abuse is thus a predominant risk factor for development of CVD.

Our study was not limited to only the correlation of ethanol abuse with impairment of lipid metabolism. It also demonstrated the impact of herbal hydro-ethanolic extract of Amorphophallus campanulatus in counter acting such health challenges due to ethanol abuse. In the present study HEEAC shows potent hypolipidemic and hypocholesterolemic activity. Among the two doses of the extract studied, 500mg/kg body wt showed better efficacy compared to the lower dose of 250mg/kg body wt. It showed significant reduction of lipid parameters over 4 weeks of study period. Not only so, the oxidative challenge that gradually developed among the ethanol treated group was also significantly reduced in the extract treated group. Lowered LPO levels and reduced kinetics of SOD, catalase and GPx are indicative of the reduced environment in the hepatocytes. Besides, the level of GSH in the extract supplemented group was comparable like that of the control group, which also provides supportive evidence that the extract has antioxidant capacity too.

In our previous study we have already demonstrated the in vitro antioxidant potency of the HEEAC as well as identified its active constituents by GC-MS analysis (23, 24). The extract showed presence of polyphenols and flavonoid fractions that may be responsible for its antioxidant property whereas presence of phytosterols may account for its hypolipidemic action.

Not only the biochemical parameters, in this study even the histological sections from the different animal groups reveal that fatty accumulation as lipid droplets is observed in hepatic tissue from ethanol treated group. However, such fatty infiltration is not found in tissue sections of animals that received the HEEAC at both doses.

Phytoconstituents and herbal extracts are gaining importance as preventive and curative alternative medicine with fewer side effects. Results of this study mainly highlight the use of phyto-extracts as promising futuristic drugs in treating lifestyle diseases.

Conflict of Interest

The researcher stated that there was no conflict of interest in the manuscript

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