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Research Article

PROTECTIVE ACTIVITY OF ETHANOLIC EXTRACT OF *AMORPHOPHALLUS CAMPANULATUS* AGAINST ETHANOL INDUCED HEPATOTOXICITY IN RATS

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ABSTRACT

Objective*:* The present study was aimed to evaluate the protective role of ethanolic extract of *Amorphophallus campanulatus* Roxb. tubers against ethanol-induced hepatotoxicity in rats.

Methods*:* Adult male albino Wistar rats, weighing 150-200g, were randomized into control and experimental group. Test group of animals were treated with ethanol (2gm/kg body wt for 30 days). Silymarin was used as a standard reference. Ethanolic extract of *Amorphophallus campanulatus* (250 mg/kg body wt and 500 mg/kg body wt) were administered to the ethanol treated rats simultaneously for thirty days. The extract at these two doses were also administered to the animals separately to study the presence of any harmful effect on the liver function.

Results*:* Administration of ethanolic extract of *Amorphophallus campanulatus* at two different doses significantly normalized the values of SOD, CAT, GSH and TBARS. The elevated serum enzymatic levels of AST, ALT, ALP and total protein were significantly restored towards normal by pre-treatment with ethanolic extract of *Amorphophallus campanulatus* ($p<0.001$).However, no significant alteration in the liver marker enzymes and the antioxidant markers in liver tissue was observed in the only extract treated groups when compared to control, indicating their non-toxic nature. There was also no significant dose dependent variation observed when treated with two different concentrations of the extract. Histopathological studies also confirmed the hepatoprotective nature of the extracts by normalizing the hepato-cellular architecture like that of normal in the treated groups when compared to the only ethanol administered group.

Conclusion*:* The results of this study strongly indicate that ethanolic extract of *Amorphophallus campanulatus* has potent hepatoprotective action against ethanol induced hepatic damage particularly by scavenging free radicals and combating oxidative stress. Further investigation can lead to the development of phytomedicines of therapeutic significance against oxidative stress.

Keywords*: Amorphophallus campanulatus,* Ethanol, Hepatoprotection, Oxidative stress, Phytomedicine

INTRODUCTION

Alcoholic beverages have been used in human societies since beginning of recorded history. The relation between alcohol consumption and health outcomes is complex and multi-dimensional [1]. Alcohol abuse and alcoholism are serious current health and socio-economic problem worldwide. Despite great progress made in this field, the development of suitable medications for the treatment of alcoholism remains a challenging goal for alcohol research.

Liver, the largest organ of the body comprising 2-3% of the total adult body weight, is primarily concerned with the metabolic activity of organisms [2]. It is also the central site for the biotransformation of xenobiotic chemicals and therefore is involved in the detoxifying mechanism of the body. Ethanol, a fat soluble non-electrolyte, after being readily absorbed from the gastrointestinal tract, diffuse rapidly into circulation and then gets distributed uniformly throughout the body [3]. Most of the ethanol consumed gets metabolized primarily by the hepatocyte alcohol dehydrogenase (ADH) enzyme and partly by the mitochondrial cytochrome P450 2E1 (CYP2E1) system. Previously it was believed that ethanol in itself is non-toxic, rather the nutritional deficiencies accompanying it were the actual cause underlying the pathogenesis. However, it has been shown by Leiber and De Carli that in rats, ethanol induced liver damage developed despite of sufficient nutrition [4].

Alcoholic liver disease (ALD) is the common consequence of prolonged and heavy alcohol intake. The most suitable reason in the prognosis of alcoholic pathogenesis lies in its course of metabolism. Ethanol metabolism also influences the redox status of liver and disruption of this redox balance causes rapid free radical generation thus resulting in a state, often termed as oxidative stress.

In traditional medical practices, followed throughout the world, herbs play a major role in the management of various disorders. The use of bioactive plant-derived compounds is on the rise, because the main drawback with the use of synthetic drugs is the side effects which can be even more dangerous than the diseases they claim to cure. Poly-herbal formulations and crude plant extracts are more preferred in the field of herbal medicine than administration of active components only. It is believed that components that are not active can act to improve the stability, bioavailability, solubility or half-life of the active components. Hence a particular active principle in pure form may exert only a fraction of its pharmacological activity that it has in the plant matrix, thus highlighting the fact that use of whole plant or plant mixtures is more effective in treating various ailments [5].

Amorphophallus campanulatus, belonging to the family of Araceae is a perennial herb commonly known as elephant foot yam. It is basically a tuber crop of South East Asian origin and is largely cultivated throughout the plains of India for using its corm (bulb) as food [6]. Moreover, the tuber is widely used in many Ayurvedic preparations, and prescribed in liver diseases, bronchitis, asthma, abdominal pain, emesis, dysentery, enlargement of spleen, piles, elephantiasis, and rheumatic swellings. These corms have been reported to possess antibacterial [7], anti-helminthic [8], anti-inflammatory [9] and cytotoxic [10] properties. So the present study was undertaken to evaluate the protective effect of *Amorphophallus campanulatus* on ethanol induced hepatic oxidative stress in rats.

MATERIALS AND METHODS

Chemicals

All reagents used in the experiment were of analytical grade and purchased either from Sigma-Aldrich, USA or Merck, India or from Hi-media Laboratories Pvt. Ltd., Mumbai, India.

Procurement of plant materials

Fresh tubers of *Amorphophallus campanulatus* (*AC*) were purchased from local Kolkata Municipal Corporation approved vegetable market, West Bengal, India. Authentication was done by Dr. Krishnendu Sarkar, Associate Professor, Department of Botany, Rammohan College under University of Calcutta, West Bengal, (India).

Preparation of Ethanolic Extract

Raw fresh tubers after procurement from local market 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 ºC, until materials became crispy in texture. This dried material was grinded into coarse powder form and stored at -4ºC in air tight Tarson jars until further use.

For the preparation of the ethanolic extract, 20 gm of the powdered dried sample was taken in the thimble of Soxhlet and extracted with 250 ml of ethanol (70%) in the round bottomed flask continuously for a week. The extract was then filtered through muslin cloth, centrifuged and the collected filtrate was evaporated to dryness using rotary evaporator (B.C. Chatterjee & Co., Kolkata, West Bengal, India). The dried sample was collected and stored in air tight plastic vials at -4ºC for future use.

Animals and diet

The present study was conducted on male albino Wistar rats weighing 150-200 grams (170 ± 2.3), in strict accordance with the recommendations of Committee for the Purpose of Control and Supervision on Experiments on Animals (CPCSEA). All surgery was performed under sodium pentobarbital anesthesia, and all efforts were made to minimize suffering. Rats were caged in Tarson's polypropylene autoclavable cages with 2-3 rats per cage, in a well ventilated room at temperatures ranging between 28°C to 32°C and 12 h light: 12 h dark cycle. Throughout the experimental period the animals had access to freshly prepared diet and water *ad libitum*. The animals were acclimatized with the laboratory environment for one week prior to the experiment initiation.

Preparation of doses and treatments

40% ethanol (2gm/kg body weight), standardized from our previous experiments [11], was administered intraperitoneally, once daily, for thirty days continuously to develop oxidative stress in rats. Silymarin at an oral dose of 100mg/kg body weight was used as standard drug control in the experiment [12]. Two different doses (250 and 500 mg/kg body weight) of ethanolic extract of AC dissolved in mammalian normal saline (0.9% NaCl) were prepared for intraperitoneal administration to the animals. It was reported that the extracts of AC tubers were safe upto the dose of 2000mg/kg body weight [13].

Experimental Design

Rats were divided into seven groups with six rats in each group and those were treated as follows for thirty consecutive days

Group I: Control, fed on standard diet and water

Group II: Negative Control, treated with 40% ethanol along with standard diet and water

Group III: Silymarin (100mg/kg body wt) + 40% ethanol treated group along with standard diet and water

Group IV: Ethanolic extract of *AC* (250 mg/kg body wt) + 40% ethanol treated group along with standard diet and water

Group V: Ethanolic extract of *AC* (500 mg/kg body wt) + 40% ethanol treated group along with standard diet and water

Group VI: Ethanolic extract of *AC* (250 mg/kg body wt) along with standard diet and water

Group VII: Ethanolic extract of *AC* (500 mg/kg body wt) along with standard diet and water

Collection of sample

After thirty days of continuous treatment, the animals were kept fasted overnight without any treatment. The day following, the animals were anesthetized and blood samples were collected by cardiac puncture. The collected blood was allowed to stand for some time to separate out the serum. Liver were excised, trimmed of connective tissues, rinsed with ice cold saline to eliminate blood contamination, dried by blotting with filter paper and weighed to obtain the organ weight. Part of the tissue was used for homogenate preparation and the other part was transferred to fixative for preparation of histological sections.

Preparation of liver homogenate

A portion of liver was weighed, perfused with saline and homogenized in chilled phosphate buffered saline (PBS) pH 7.4 using tissue grinder with power drive homogenizer (REMI, Vasai, India). The homogenates were centrifuged at 800 g for 5 min at 4°C to separate the nuclear debris. The supernatant so obtained was centrifuged at 10,500 g for 20 min at 4°C to get the post mitochondrial supernatant (PMS) which was used to assay superoxide dismutase (SOD) [14], catalase [15], reduced glutathione (GSH) [16] and thiobarbituric acid reactive substances (TBARS) [17].

Estimation of liver marker enzymes

Serum aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP) were estimated using commercially available kit reagents manufactured by Span Diagnostics Ltd, India. Serum and tissue protein was estimated by the method of Lowry *et al.,* [18]. Absorbances at different wavelengths were measured using UV- Vis Single Beam Spectrophotometer (Systronics 118).

Histopathological Study

The buffered formal fixed tissues were paraffin embedded after undergoing proper dehydration and clearing steps. Tissue blocks were mounted on rotary microtome to obtain sections of 5 µm thickness. The sections were then used for haematoxyline-eosin staining [19] to detect changes in liver morphology.

Statistical Analysis

Results were presented as mean ± standard error of mean (S.E.M). Statistical significance between the control groups and the test groups were analyzed by one way Analysis of Variance (ANOVA) after ascertaining homogeneity of variance, followed by Tucky's Kramer post-hoc analysis using Graphpad InStat Software version 3.10.

RESULTS

Effect on relative liver weight

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 when compared to control. Consequently, simultaneous treatment with ethanolic extract of *AC* (250 and 500 mg/kg body weight) and silymarin (100mg/kg body weight) along with ethanol showed significant decrease ($p<0.001$) in liver weight and ratio of liver weight to body weight as compared to the ethanol treated group. No significant change in liver weight and ratio of liver weight to body weight was observed among the only extract and silymarin treated groups when compared to control. Moreover, no significant change was also observed among the two different doses of extract treated groups. The results are presented in Table 1.

Effect on liver marker enzymes

Table 2 shows the effect of ethanol and subsequent treatment with ethanolic extract of AC and the standard hepatoprotective drug, silymarin on the levels of serum liver marker enzymes. Single dose of ethanol/day significantly ($p<0.001$) increased the levels of AST, ALT and ALP while decreased the levels of total protein when compared with the control group. On the contrary, co-administration of ethanol along with ethanolic extract of AC at 250 and 500 mg/kg body weight showed significant reduction ($p<0.001$) in the levels of AST, ALT and ALP while elevation in total protein level when compared with the ethanol treated group. The results of liver marker enzymes in the extract co-administered group were comparable with that of the silymarin treated group. It is worth mentioning that no significant changes in the levels of AST, ALT, ALP and total protein were observed among group VI and VII when compared with control, indicating the absence of any harmful effect of the extract on liver.

Effect on Antioxidant Status

Table 3 shows the changes in the antioxidant status of liver tissue among the different groups. The levels of reduced glutathione (GSH), activity of superoxide dismutase (SOD) and catalase (CAT) significantly ($p<0.001$) decreased in the ethanol treated group in comparison to control. However, the decrease in these important non-enzymic and enzymic antioxidants were arrested significantly in the groups that received extracts (250 and 500 mg/kg) simultaneously with ethanol. On the other hand, the level of TBARS as an indicator of the extent of lipid peroxidation in the tissues was significantly elevated ($p<0.001$) in the ethanol treated group in comparison to control. This was also attenuated significantly on simultaneous treatment with extract of AC prior to ethanol administration. It is worth mentioning that the changes in the levels of these tissue antioxidant status markers showed no significant difference among the two different concentration of extract treated groups and the results were comparable with that of the silymarin treated group. No significant alteration in the above parameters were also observed among the only extract treated and control groups.

Table1: Effect of ethanolic extract of *AC* on liver weight and liver wt to body wt ratio in the control and experimental rats.

Groups	Final Body Weight (gm)	Liver Weight (gm)	Liver Wt to Body Wt Ratio (%)
Control	175.00 ± 0.02	5.6 ± 0.05	3.20 ± 0.18
Only Ethanol	165.00 ± 0.19	6.9 ± 0.08*	4.18 ± 0.11*
Ethanol+Silymarin	170.00 ± 0.16	5.5 ± 0.14**	3.29 ± 0.06**
Ethanol+*AC* (250mg/kg BW)	168.00 ± 0.08	5.6 ± 0.24** ns	3.21 ± 0.17** ns
Ethanol+*AC* (500mg/kg BW)	170.00 ± 0.22	5.8 ± 0.18**	3.35 ± 0.13**
Only *AC* (250mg/kg BW)	172.00 ± 0.42	5.6 ± 0.22	3.37 ± 0.08
Only *AC* (500 mg/kg BW)	171.00 ± 0.09	5.7 ± 0.17	3.45 ± 0.11

All values are expressed as MEAN ± SEM, of 6 animals in each group. Data were 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. ethanol + *AC* (500mg/kg BW) , **P<0.001 vs. only ethanol

Table2: Effect of ethanolic extract of *AC* on the levels of ALT, AST, ALP and Total Protein in the control and experimental rats.

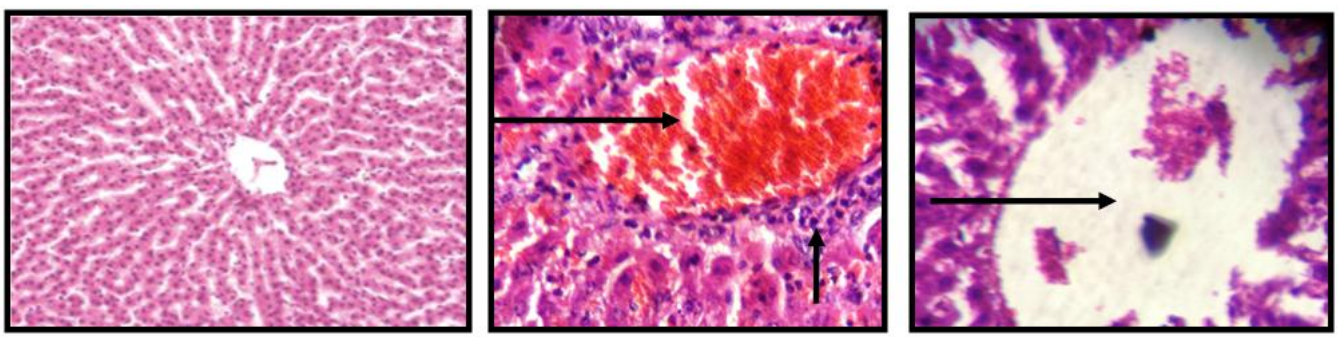
Groups	ALT (U/L)	AST (U/L)	ALP (U/L)	Total Protein (gm/dl)
Control	50.79 ± 2.9	192.59 ± 7.5	185.64 ± 2.8	7.5 ± 0.15
Only Ethanol	109.19 ± 3.4*	379.35 ± 3.1*	357.92 ± 3.9*	6.3 ± 0.21*
Ethanol+Silymarin	61.48 ± 1.7**	212.49 ± 4.4**	243.68 ± 4.3**	7.3 ± 0.12**
Ethanol+*AC* (250mg/kg BW)	79.82 ± 2.3** ns	254.33 ± 4.0** ns	278.43 ± 6.0** ns	7.6 ± 0.13** ns
Ethanol+*AC* (500mg/kg BW)	71.88 ± 1.6**	242.09 ± 3.4**	265.59 ± 5.0**	7.7 ± 0.13**
Only *AC* (250mg/kg BW)	63.52 ± 2.5	207.96 ± 4.9	204.88 ± 4.0	7.4 ± 0.13
Only *AC* (500 mg/kg BW)	68.96 ± 2.0	210.04 ± 4.0	202.45 ± 3.5	7.0 ± 0.10

All values are expressed as MEAN ± SEM, of 6 animals in each group. Data were 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. ethanol + *AC* (500mg/kg BW) , **P<0.001 vs. only ethanol

Table3: Effect of ethanolic extract of *AC* on the levels of TBARS, GSH and activities of antioxidant enzymes (SOD and CAT) from hepatic tissue of control and experimental rats.

Groups	GSH (mg/100 gm)	SOD (U/min/mg)	CAT (µmol H_2O_2/min/mg)	TBARS in terms of MDA (µmol/100 gm)
Control	81.22 ± 1.9	3.62 ± 0.07	111.41 ± 1.2	24.65 ± 2.2
Only Ethanol	46.84 ± 1.8*	1.61 ± 0.1*	84.04 ± 0.9*	52.71 ± 2.4*
Ethanol+Silymarin	77.18 ± 0.8**	3.06 ± 0.08**	104.27 ± 2.3**	26.22 ± 1.3**
Ethanol+*AC* (250mg/kg BW)	70.85 ± 2.5** ns	2.49± 0.04** ns	94.25 ± 2.3*** ns	34.72 ± 1.5** ns
Ethanol+*AC* (500mg/kg BW)	72.88 ± 2.6**	2.62 ± 0.06**	100.03 ± 0.8**	33.71 ± 1.5**
Only *AC* (250mg/kg BW)	75.88 ± 1.8	3.05 ± 0.05	103.18 ± 2.9	26.50 ± 1.9
Only *AC* (500 mg/kg BW)	70.67 ± 2.3	2.85 ± 0.1	101.35 ± 1.9	31.36 ± 1.7

All values are expressed as MEAN ± SEM of 6 animals in each group. Data were 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. ethanol + *AC* (500mg/kg BW), **P<0.001 vs. only ethanol, *** P<0.05 vs. only ethanol



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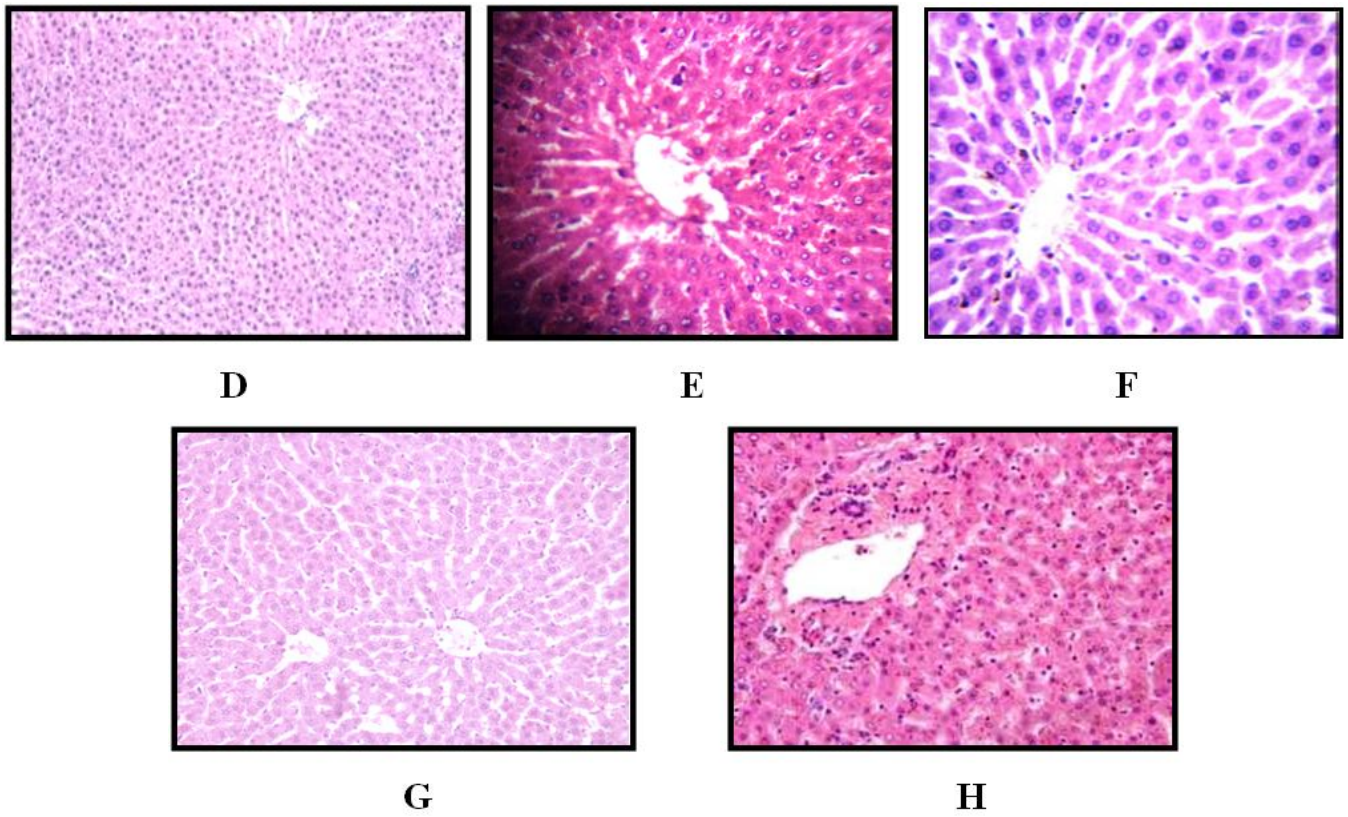

Fig. 1: Representative Images of Liver Sections from control and different experimental groups of animals in the study (Haematoxylin-Eosin Staining)

A: Liver section from control group showing normal hepatocyte architecture.

B & C: Liver section from ethanol treated group. Black arrows indicate dialatation and congestion of central vein along with cellular infiltration and necrosis.

D, E & F: Liver section from Silymarin, AC 250mg/kg body wt and AC 500mg/kg body wt simultaneously with ethanol, respectively. The sections show almost normal architecture with absence of central vein congestion, cellular infiltration and necrosis.

G & H: Liver sections from only AC 250mg/kg body wt and AC 500mg/kg body wt, respectively, showing normal architecture like that of control group.

Effect on Histopathology

The liver section from the control group showed normal lobular architecture with central veins and radiating hepatocytes (Fig 1A). Liver from rats treated with only ethanolic extract of *AC* at 250 and 500 mg/kg body weight and silymarin also exhibited near normal architecture indicating their non-toxic effect (Fig 1D, G & H). However, rats treated with ethanol alone showed marked alteration in the architecture and clear signs of liver injury indicated by severe dilatation and congestion of blood sinusoids, necrosis and cellular infiltration (Fig1B & C). Using the ethanolic extract of *AC* as protective agent against ethanol induced toxicity, a remarkable effect was observed in terms of decreased portal congestion, dilatation and cellular infiltration, as comparable to the silymarin treated group (Fig 1 E & F).

DISCUSSION

Alcohol-induced oxidative stress is linked to the metabolism of ethanol. Three metabolic pathways of ethanol have so far been described in the human body, which involve the following enzymes: alcohol dehydrogenase, microsomal ethanol oxidation system (MEOS) and catalase. Each of these pathways can produce free radicals that affect the antioxidant system. Antioxidants are known to protect against Reactive Oxygen Species (ROS) toxicity by the prevention of ROS formation, interruption of ROS attack, scavenging the reactive metabolites and converting them to less reactive molecules.

Liver being the major site for detoxification of chemical substances in the blood gets exposed to high concentrations of toxicants and toxic metabolites thus making it susceptible to injury [20]. However, in this present study it is highlighted that liver, the prime target for ethanol metabolism gets severely affected during chronic ethanol exposure. Ethanol itself forms a toxic environment favorable to oxidative stress such as hypoxia, endotoxaemia and cytokines release [21].The toxic metabolic effects of ethanol oxidation are mainly due to increased liberation of ROS, production of deleterious active acetaldehyde, increased "NADH/NAD" ratio and disturbed intracellular calcium stores [22]. Increased NADH production leads to elevated expression of xanthine oxidase, which augments superoxide production [23]. Free radicals have been implicated in alcoholic liver disease in various ways. Mechanisms that are thought to be involved are impairment of antioxidant defenses, as well as production of reactive oxygen species (ROS) like hydroxyl, superoxide and hydrogen peroxide [24-26]. These reactive oxygen species may, in turn, lead to elevated levels of lipid peroxidation products, resulting in the formation of adduct with cellular proteins and hence limiting their function. Adducts may also be formed with nucleic acids which eventually leads to cellular damage.

An obvious sign of hepatic injury is leakage of cellular enzymes into serum, and measurement of the activity levels of enzymes in the body fluids is a useful monitor of a disease state. In our present study, it is observed that the levels of hepatic marker enzymes, ALT, AST and ALP are significantly elevated in the ethanol treated group along with decrease in total serum protein. The elevated levels of

serum marker enzymes are indicative of cellular leakage and loss of functional integrity of cellular membrane in liver [27]. Moreover, decline in total protein content can also be deemed as a useful index of the severity of cellular dysfunction in chronic liver disease. This study shows that simultaneous treatment with ethanolic extract of *AC* at dose of 250 and 500 mg/kg body weight along with ethanol, significantly lowers the elevated levels of the liver marker enzymes in serum almost like that of silymarin treated group. Restoration of the enzyme levels near to their respective control values clearly indicates that the tuber extract stabilizes the plasma membrane and helps in healing of the hepatic tissues. Not only so, restoration of total protein level by stimulating protein synthesis in the extract treated group is an important feature of the hepatoprotective nature of the extract. This accelerates the regeneration process and production of new liver cells [28].

Disruption of balance between prooxidant and antioxidant in chronic ethanol ingestion leads to generation of free radical cloud that alters normal functioning of the cells by damaging cellular macromolecules. In this context, it has already been reported that lipid peroxidation is the most prominent destructive process in ethanol induced liver injury [29]. Our study supports this phenomenon by showing the elevated levels of TBARS in tissue homogenates of ethanol treated rats. TBARS are mainly unstable peroxides, and are by-products of membrane phospholipid peroxidation. Increased concentration of TBARS is thus indicative of enhanced lipid peroxidation and failure of the antioxidant defense mechanism to inhibit free radical generation. Compromise of the body's self antioxidant capacity is also manifested by decrease in the levels of reduced glutathione (GSH), most abundant naturally occurring tripeptide and non-enzymatic cellular antioxidant, in the ethanol treated group. GSH is mainly concerned with the removal of free radicals like superoxide, H_2O_2, hydroxyl, maintenance of membrane protein, detoxification of foreign chemicals and biotransformation of drugs [30]. Therefore, depletion of GSH content from hepatic tissue in the ethanol treated group highlights the altered redox balance in hepatic tissue as a result of chronic ethanol administration. However, on administration of ethanolic extract of *AC* (250 and 500mg/kg body wt), thirty minutes prior to ethanol, showed significant protective effect by reducing the levels of TBARS and attenuating GSH depletion from liver tissue.

Serum activities of superoxide dismutase (SOD) and catalase (CAT) are the most sensitive enzymatic index in liver injury caused by ROS and oxidative stress. SOD is one of the most abundant intracellular antioxidant enzymes present in all aerobic cells and it has an antitoxic effect against ROS [31]. CAT on the other hand is a haemoprotein and it protects the cell from the accumulation of H_2O_2 by dismutating it to form H_2O and O_2 [32]. Therefore, reduction in the activities of these enzymes may indicate the toxic effects of ROS produced by toxicants. In this study ethanol decreases the activities of SOD and CAT in liver tissue. However, the situation becomes reversed on treatment with ethanolic extract of *AC* prior to ethanol administration. This suggests that the ethanolic extract of *Amorphophallus campanulatus* at both concentrations (250 and 500mg/kg body weight) shows significant ROS scavenging activity with lesser oxidative damage to the hepatocytes. No significant dose dependent variation in the protective action is observed between the two doses. The potent hepatoprotective activity of this extract is clearly manifested by its ability to restore the levels of liver marker enzymes, attenuate the depletion of GSH, production of TBARS and maintain the activities of SOD and CAT in liver tissue. Moreover, on treatment with the extract the hepatocyte morphology is also restored to normal as examined by histopathological staining techniques.

Preliminary phytochemical screening of the ethanolic extract of *Amorphophallus campanulatus* reveals the presence of phenols and flavonoids in it. Besides, the extract also shows potent ability to scavenge free radicals like superoxide, hydroxyl radical generated by in vitro mechanisms, inhibit lipid peroxidation, DPPH radical scavenging and reducing power [33]. So, the results of the present *in vivo* study are well in accordance with the above mentioned *in vitro* observation, where the antioxidant property of this tuber has already been established. This is also worth mentioning that though the *in vivo* antioxidant property of the extract of *Amorphophallus campanulatus* against CCl_4 [13] and Acetaminophen [34] induced hepatotoxicity has already been reported, their hepatoprotective property against ethanol induced liver injury is being reported for the first time in this study.

In conclusion, it can be said that the ethanolic extract of *Amorphophallus campanulatus* provides protection to hepatic tissue of rat against ethanol induced oxidative stress probably through its antioxidant action. Results of this study further leads to the need for thorough investigation in terms of active component identification and the probable mechanism of action, which is beyond the scope of the present study. The results presented here may be of future therapeutic significance particularly where man himself chronically exposes to ethanol as his addictive habit. As *Amorphophallus campanulatus* is a very popular, low cost tuberous vegetable and forms a part of the regular diet among the East Indian population, it may also serve as a possible nutritional intervention and the extract itself or fractions obtained there from may be used as a future nutritional supplement to combat oxidative stress-induced tissue damage.

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