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J. Nat. Prod. Plant Resour., 2013, 3 (2):76-79
(http://scholarsresearchlibrary.com/archive.html)

ISSN : 2231 – 3184
CODEN (USA): JNPPB7

Hepatoprotective activity of *Cayratia carnosa* on liver damage caused by lead acetate in rats

Suganthi. V.*, Gowri. S and K. Gurusamy

P.G. Department of Biochemistry, Dr. N. G. P. Arts and Science College, Coimbatore

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ABSTRACT

The objective of this study was to investigate the hepatoprotective activity of methanol extract of leaves of Cayratia carnosa (Mecc) against lead acetate induced hepatotoxicity. The material was shade dried; they were powdered and extracted with methanol. The hepatoprotective activity of methanol extract was assessed in lead acetate induced hepatotoxic rats. Alteration in the levels of biochemical markers of hepatic damage like ALT, AST, ALP, ACP and LDH and serum parameters like Cholesterol, triglycerides and bilirubin were tested in both lead acetate treated and untreated groups. Lead acetate induced group has enhanced the ALT, AST, ALP, ACP and LDH and serum parameters like Cholesterol, triglycerides and bilirubin in liver. Treatment with methanolic extract of Cayratia carnosa leaves (250 mg/kg/bwt) has brought back the alerted levels of biochemical markers to near normal levels in the dose dependent manner. Our findings suggested that methanol leaf extract of Cayratia carnosa possessed hepato protective activity.

Keywords: *Cayratia carnosa* , lead acetate , hepatoprotective.

INTRODUCTION

Liver is one of the largest Organs in human body and the chief site for intense metabolism and excretion. So it has surprising role in the maintenance, Performance and regulating homeostasis of the body. It is involved with almost all the biochemical pathways to growth, fight against disease, nutrient supply, energy provision and reproduction [1]. The major functions of the liver are carbohydrate, protein and fat metabolism detoxification, secretion of bile and storage of vitamins. Thus to maintain a healthy liver is a crucial factor for overall health & well being. [2]. Liver diseases are mainly caused by excess consumption of alcohol, toxic chemicals, infection and autoimmune disorders. Most of the hepatotoxic chemicals damage liver cells mainly by including lipid peroxidation and other oxidative damage [3]. Thus liver diseases are some of the fatal disease in the world today. They pose a serious challenge to international public health. Modern medicines have little to offer for alleviation of hepatic disease and it is chiefly the plant based preparations which are employed for their treatment of liver disorders. But there are not much drug available for the treatment of liver disorders [4,5] .

There fore many folk remedies from plant origin are tested for its potential antioxidant and hepatoprotective liver damage in experimental model. Lead acetate induced hepatotoxicity model is used for the study of hepatoprotective effect of drugs and plant extract [6].Lead is a heavy metal that can be toxic when introduced into the human and

animal bodies by ingestion or inhalation in sufficient quantities. It causes various destructive effects [7]. In human, increased levels of lead causes many serious diseases and dysfunction of organs [8,9].

The plant *Cayratia carnosa* is a traditional medicinal herb belonging to the family "Vitacea" found thought out the hotter parts of India. It is a perennial climber with stem woody at base. Ethano medical review of plant revealed that the entire plants showed astringent and anti diuretic activity. It purifies the blood was given cardiac disorders [10]. It is used for the tumor, liver disorders [11].

MATERIALS AND METHODS

Collection of Plant material

Plant materials are collected from the local area of the Kerala.

Preparation of Crude extract

The collected plant materials are shade dried and powered and macerated in 80% aqueous- Methanol for 48 hours with occasional shaking. The extract was filtered and concentrated to dark brown residue under reduced pressure on a rotary evaporator, with an approximate yield of 8%.

Animals

Swiss albino male rats [weighing 100-150g] obtained from Government Veterinary college Mannuthi, Kerala, India were used for the study. The animals were housed in standard conditions with natural light and dark cycle. They were fed with standard pellet diet and water ad libitum. Animals were acclimatized to their environment for one week prior to experimentation. All the animals were performed after approval from the Institution of Ethical Committee (IACE) (KMCRET/M.phil/03/2011) and in accordance with the recommendation for the proper care and use of laboratory.

Experimental design

Hepatic injury was induced in rats by intraperitoneal administration of single dose of lead acetate (20mg/Kg/bwt/ip). Silymarin, a known hepatoprotective agent was used as reference standard. Animals were grouped as follows.

Group I: Served as control Group II: Rats administered lead acetate (20mg/kg/bw/ip). As a single dose. Group III: Lead acetate induced rats received methanolic extract of *Cayratia carnosa* (250mg/kg/bwt) for 14 days. Group IV: Lead acetate induced rats received standarted drug Silymarin (25mg/kg/bwt) for 14 days. Group V: Control rats received methanolic extract of *Cayratia carnosa* (250mg/kg/bwt) for 14 days.

At the end of the experiment animals was sacrificed by cervical decapitation. And blood samples of each animal were collected in Sterile Centrifuge tubes allowed to clot. Serum was separated and analyzed various biochemical parameters. Like Alanine aminotransferase (ALT), Aspartate amino transferase (AST), Alkaline phosphatase (ALP), Acid phosphatase (ACP) and LDH [12,13]), Bilrubin [14] Cholesterol And Triglycerides [15].

Statistical analysis

All the values are expressed as mean ± SD. The results were analyzed statistically by Analysis of variance (ANOVA) followed by post-hoc test (e.g Tukey's HSD,Scheffe,S-N-K,Bonferroni,Duncan or Tamhan's) p values <0.005 were considered significant.

RESULTS

Results in Table I and Table II revealed a significant ($P<0.005$) elevation of serum AST, ALT, ACP, ALP , LDH , Cholesterol, Triglycerides and bilirubin levels in lead acetate treated group as compared to control group indicating that lead acetate induced damage to the hepatic cells. A significant ($P<0.005$) reduction was observed in liver marker enzymes and lipid profile levels and bilirubin in the groups treated with methanol extract of *Cayratia carnosa* in comparison with those observed in lead acetate treated group , through the decrease was maximum ($P<0.005$) in group rats which received a dose of 250 mg/kg/bwt of the methanol extract of *Cayratia carnosa* . These results suggested the protection against liver injury upon lead acetate induction.

TABLE 1: Effect of *Cayratia carnosa* leaf extract on lead acetate induced hepatic damage of liver marker enzymes in serum of control and experimental animals.

Parameters	Group I control	Group II Lead acetate	Group III Lead aceate+*MeCC*	Group IV Lead aceatte+silymarin	Group V *MeCC*
AST(U/I)	18.12± 2.50	37.05 ± 0.90[a]	25.12±0.19[b]	2 2.15±0.52[c]	19.05 ± 0.59[d]
ALT(U/I)	30.18 ± 0.62	72.54 ± 0.14[a]	45.19±0.73[b]	39.56 ± 0.89[c]	30.27 ± 0.18[d]
LDH(U/I)	145.5 ± 1.03	335.38 ± 4.50[a]	220.01±0.98[b]	150.26 ± 2.9[c]	146.16 ± 0.07[d]
ACP(U/I)	4.80 ± 0.41	11.46 ± 0.79[a]	14.11±1.24[b]	4.67 ± 0.32[c]	5.61±0.40[d]
ALP(U/I)	16.12 ± 0.19	96.90± 2.14[a]	33.52 ± 1.59[b]	18.14 ± 2.50[c]	16.16±0.81[d]

Values are expressed as mean ±SD (n=6).

Statistical comparison **Statistical significance p<0.005**

a – Group II compare with Group I b- Group III compares with Group II

c- Group IV compares with Group III d- Group V compares with Group I

TABLE 2: Effect of *Cayratia carnosa* leaf extract on lead acetate induced hepatic damage in serum of cholesterol, triglyceride and bilirubin in control and experimental animals.

Parameters	Group I Control	Group II Lead acetate induced	Group III Lead acetate +*MeCC*	Group IV Lead aceate+silymarin	Group V *MeCC* alone
Cholesterol(mg/dl)	108.66 ± 3.48	229.75± 0.75[a]	127.66±2.15[b]	110.17 ± 1.94[c]	109.16 ± 4.94[d]
Triglycerides(mg/dl)	58.4 ± 2.9	99.42 ± 5.2[a]	60.6±4.7[b]	59.12± 5.2[c]	58.72 ± 3.4[d]
Bilirubin(mg/dl)	0.51±0.002	1.72±0.03[a]	0.46±0.02[b]	0.431±0.002[c]	0.5±0.03[d]

Values are expressed as mean ±SD (n=6).
Statistical comparisons are similar to that of the above table

DISCUSSION

Lead is well known for its involvement in various biochemical and metabolic process. Absorption of inorganic lead can lead to certain biochemical and metabolic toxicities [16]. Lead is known to inhibit many enzyme activities [17]. It is widely used for inducing experimental hepatic damage due to free radical formation during it's metabolism by hepatic microsome leading to lipid peroxidation and consequently liver damage.

Hepato cellular necrosis leads to very high levels of AST, ALT in blood released from liver. Between the two alanine is a better index of injury represents 90 % of total enzymes present in the body. The normalization of AST, ALT in plant treated groups indicated the stabilization of plasma membrane and protection of hepatocytes against the damage caused by lead acetate [18]. The elevated levels of serum marker enzymes are inactive of cellular leakage and loss of functional integrity of cellular membrane in liver. In the assessment of liver damage by lead acetate the determination of liver marker enzyme such as AST, ALT is often used. In necrosis or membrane damage the enzymes are released into circulation and it can be there for meseaured in serum as marker of hepatic damage[19].

ACP is a marker enzyme of the lysosomal membrane. Increased ACP activities could result in indiscriminate hydrolysis of phosphate esters which are potential energy source for the cell and this may constitute a possible threat to the well being of the liver which may result in cell death. ALP is a marker enzyme of the plasma membrane and endoplasmic reticulum. It is used to assess the integrity of the plasma membrane. The reduction in the ALP activity in the male rat liver might be adduced to either loss of membrane components into the extracellular fluid in the serum inactivation of the enzyme molecule or inhibition of the enzyme activity at the cellular / molecular level [20]

ALP activites on other hand are related to functioning of hepatocytes, it's increase in serum is due to increased synthesis in the presence of increased biliary pressure. The rise in serum ALP activity may be attributed to the disturbance in the secretary activity or in the transport of metabolites or may be due to altered syntheses certain enzymes as in other hepato toxic condition. This effect is in agreement with the commonly accepted view that serum levels of transaminases return to normal with the healing of hepatic parenchyma and regeneration of hepatocytes.

Serum ALP and total bilirubin levels are also related to the status and function of hepatic cells, increase in serum ALP is due to increased synthesis in presence of biliary pressure [21].

Bilirubin has been used to evaluate chemically induced hepatic injury. It is one of the most useful clinical clues to the severity of necrosis and its accumulation is a measure of binding, conjugation, and execratory capacity of hepatocytes. Decrease in serum bilirubin after treatment with the extract in liver damage induced by lead acetate indicate the effectiveness of the extract in normal function status of the liver [22].

Lipid profile such as cholesterol, triglycerides are increased in hepatopathy . In body cholesterol exist in two form free cholesterol and estrified form. The lipid content of hepatocytes is regulated by the integrated activities of cellular enzymes that catalyze lipid uptake, synthesis, oxidation and export. Fat accumulates within the hepatocytes when the "input" (either uptake or synthesis) of fatty acids to hepatocytes exceeds their "output" (oxidation and export). Different factors, extrahepatic and intrahepatic, can impair both regulator mechanisms and, therefore, promote triglyceride and cholesterol accumulation in the liver of rats with short-term and long-term (1 year) pre-hepatic portal hypertension [23].

CONCLUSION

Thus the present study revealed that *Cayratia carnosa* possesses significant hepatoprotective activity. However further studies on other models and clinical trails are required to confirm these results and to establish the exact mechanism of action and active principle involved in hepatoprotective effect.

Acknowledgements
The authors gratefully acknowledged the valuable contributions of the management of Dr. N.G.P Arts and Science College, Coimbatore.

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