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RESEARCH ARTICLE

HEPATO PROTECTIVE AND ANTIOXIDANT ACTIVITY OF *Mimosa pudica* ON CARBON TETRA CHLORIDE –INDUCED HEPATIC DAMAGE IN RATS

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ABSTRACT

The ethanol extract of *Mimosa pudica* (Mimosaceae) leaves was evaluated for its hepatoprotective and antioxidant activities against carbon tetrachloride (CCl4)-induced liver damage, in wistar albino rats. The ethanol extract of *Mimosa pudica* (Mimosaceae) leaves (200 mg/kg body weight, p.o.) was administered to the experimental rats for 14 days. Silymarin (50 mg/kg body weight (b.w.) was given as the standard drug. The hepatoprotective activity was assessed using various serum biochemical parameters as glutamate oxaloacetate transaminase (SGOT), glutamate pyruvate transaminase (SGPT), alkaline phosphatase (ALP), bilirubin, and total proteins. Malondialdehyde (MDA) level as well as the activities of superoxide dismutase (SOD), reduced glutathione (GSH) and catalase (CAT) was determined to explain the possible mechanism of activity. The substantially elevated levels of serum GOT, GPT, ALP and total bilirubin, due to CCl4 treatment, were restored towards near normal by *Mimosa pudica* (Mimosaceae), in a dose. *Mimosa pudica* (Mimosaceae) also increased the serum total proteins of CCl4-intoxicated rats. Reduced enzymatic and non-enzymatic antioxidant levels and elevated lipid peroxide levels were restored towards near normal, by administration of *Mimosa pudica* (Mimosaceae). The results revealed that, the ethanol extract of *Mimosa pudica* (Mimosaceae) afforded significant dose dependent hepatoprotective and antioxidant effects in CCl4-induced hepatic damage.

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INTRODUCTION

Liver is the most important organ of metabolism, synthesize, secretion, detoxification, storage and excretion. Therefore, the maintenance of a healthy liver is vital to the overall health and well being. The impairment of the liver function is generally caused by xenobiotics, toxic chemicals, infections and autoimmune disorders. Excess production of reactive oxygen species and /or defective cellular antioxidant system have been widely implicated in the pathogenecity of chronic liver injury (Bataller and Brenner, 2005). CCl4-inducedhepatotoxicity model is frequently used for the investigation of hepatoprotective effect of drugs and plant extracts (Chaudhari *et al.,* 2009). The major cause ofCCl4-

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induced hepatic damage is due to generation of free radicals followed by lipid per oxidation and decreased activities of antioxidant enzyme levels (Aghel *et al.,* 2007). The resulting hepatic injury was characterized by the leakage of cellular enzymes into the blood stream and centrilobular necrosis (Muriel *et al.,*2001). Antioxidant action has been reported to play a crucial role in the hepatoprotective capacity of many plants, such as *Strychnos potatorum* (Sanmugapriya and Venkataraman, 2006), *Curculigo orchioides* (Venukumar and Latha, 2002), *Rosa domascena* (Achuthan *et al.,* 2003), and *Chamomile capitula*(Gupta and Misra, 2006). Several Indian medicinal plants have been extensively used in the Indian traditional system of medicine for the treatment of hepatic disorders. Thus search for the crude drugs of plant origin with antioxidant activity has been a central focus of study of hepato protection.

Mimosa pudica (Mimosaceae) known as chue Mue, is a stout stragling prostrate shrubby plant with the compound leaves which gets sensitive on touching, pinousstipules and globose pinkish flower heads, grows as weed in almost all parts of the country (Ghani, 2003). Leaves and stems of the plant have been reported to contain analkaloid mimosine, leaves also contain mucilage and root contains tannins (Ghani, 2003). *Mimosa pudica* is used for its anti-hyperglycemic (Uma maheswari, 2007), antidiarrhoeal (Balakrishnan, *et al.,* 2006), anti-convulsant (Bum, *et al.,* 2004) and cytotoxic properties (Sadia Afreen Chowdhury, *et al.,*2008). The plant also contains turgorins, leaves and roots are used in treatment of piles and fistula. Paste of leaves is applied to hydrocele. Cotton impregnated with juice of leaves is used for dressing sinus. Plant is also used in the treatment of sore gum and is used as a blood purifier (Ghani, 2003). The present study reports the anti hepato protective activity of an ethanolic extracts of *Mimosa pudica* in CCl4 Induced liver injury in rats.

MATERIALS AND METHODS

***Plant material*:** The leaves of *Mimosa pudica* were procured from the Pattukkottai in the month of Febuary-2010. The shade dried coarse powder bark of *Mimosa pudica* (500 g) was packed in the Sox let apparatus and extracted with 1.5 liters of 95% ethanol at a temperature of 40-50°C for 72 h. The extract was filtered and then concentrated to dryness in a rotary evaporator under reduced pressure at a temperature of 40°C.The resultant black colored residue was stored in a desiccators for use in subsequent experiments and considered as the crude ethanol extract. The yield of the extract was 11% w/w. The preliminary photochemical screening was performed for the extract (Horbone, 1998; Kokate *et al.,* 1998; Trease and Evans, 2000). .

Preliminary Phytochemical Analysis: The ethanolic extract was then subjected to preliminary phyto chemical (Harbone, 1984) analysis to assess the presence of various phyto constituents, it revealed the presence of flavonoids, alkaloids and glycosides. Preliminary Thin layer chromatography studies also confirmed these constituents (Wagner and Blatt, 1996)

Acute oral toxicity study

Acute oral toxicity study was performed as per OECD-423 guidelines (Ecobichon, 1997). Female Swiss albino mice (20-25 g) were randomly distributed to four groups of six each. The animals were fasted overnight and the drug was administered orally at the doses of 100, 200, 400, 800, 1600, and 3200mg/kg b.w. The animals were closely observed for the first 24 h for any toxic symptoms and for 72 h for any mortality rate.

Hepatoprotective studies

Experimental design

Rats were fasted for 16 h and divided in to five groups of six each. Group I(normal control), was given normal saline orally at a dose of 5 ml/kg b.w. GroupII (hepatotoxic control), received CCl4 at a dose of 2 ml/kg b.w. sub cutaneously.Group III received *Mimosa pudica* (Mimosaceae) orally at a dose of 200 mg /kg b.w,respectively and CCl4-induction. and IV received silymarin orally at a dose of 50 mg/kg b.w and CCl4-induction The extract and standard drug silymarin were administered orally, once in a day for 14 days, and the dose of CCl4 (2 ml/kg, 1: 1dilution with liquid

paraffin) (Lin *et al.,* 1998) was administered subcutaneously at the lower abdomen to groups II to IV, after every 72 h. After 24 h of the last administration of the extract and in 18 h fasting condition, the blood was collected from all animals by retro-orbital bleeding, using micro capillary technique. Serum was separated and used for the determination of biochemical parameters, such as serum glutamate oxaloacetate transaminase (SGOT), serum glutamine pyruvate transaminase (SGPT), serum alkaline phosphatase (SALP), bilirubin, and total proteins (using Automated Span Diagnostic Reagents, Mumbai, India).

Assessment of antioxidant activity

After the collection of blood samples, the rats were sacrificed and their livers were excised, rinsed in ice-cold normal saline (pH 7.4), blotted dry, and weighed. A 10% w/v of the homogenate was prepared in 1.15% KCl and processed for the estimation of lipid peroxidation and reduced glutathione content, super oxide dismutase (SOD), catalase (CAT), and the total proteins were estimated.

Estimation of Lipid Peroxidation (LPO)

The level of TBARS (Thiobarbituric acid reactive substances) in the liver was measured (Ohkawa *et al.,*1979). A mixture of 0.4 ml of 10% liver homogenate, 1.5 ml of 8.1% sodium dodecyl sulphate (SDS), 1.5 ml of 20% acetate buffer (pH3.5) and 1.5 ml of 0.8% of TBA solution was heated at 95°C for 1h. After cooling, 5.0 ml of *n*-butanol-pyridine (15:1) was added and the absorbance of the butanol-pyridine layer was measured at 532 nm.

Estimation of Reduced Glutathione (GSH)

Glutathione was measured according to the method of Ellman (1959). The equal quantity of homogenate was mixed with 10% trichloroacetic acid and centrifuged to separate the proteins. To 0.01 ml of this supernatant, 2 ml of 0.2 mM, phosphate buffer (pH 8.4), 0.5 ml of 0.6 mM, 5'5-dithio, bis (2-nitrobenzoic acid) and 0.4 ml double distilled water was added. Mixture was vortexed and the absorbance read at 412 nm within 15 min. The concentration of glutathione was expressed as µg/mg of protein

Estimation of Super oxide Dismutase (SOD)

The activity of SOD in tissue was assayed by the method of Kakkar *et al.,*(1984).1ml of nitroblue tetrazolium (NBT) solution (156µg NBT in 100 mM Phosphate buffer, pH7.4), 1ml NADH solution (468µg NADH in 100 mM phosphate buffer,pH7.4) and 0.1ml of liver homogenate were mixed. The reaction started by adding100µg of phenazine metho sulphate (PMS) solution (60µg PMS in 100mM phosphate buffer, pH 7.4) to the mixture. The reaction mixture was incubated at25°C for 5 min and the absorbance at 560nm was measured against blank sample without liver homogenate.

Estimation of Catalase (CAT)

Catalase activity was measured according to the method by Aebi *et al.* (1974).The estimation was done spectrophotometrically following the decrease in absorbance at 230 nm. The tissue was homogenized in M/150 phosphate buffer (pH 7.0) at 1-4°C and centrifuged at 5000 rpm. The reaction mixture contained0.01M phosphate buffer (7.0), 30mM H2O2 and the enzyme extract. The specific activity of catalase was expressed in terms of units/mg protein. Absorbance values were compared with a standard curve generated from known CAT.

Statistical analysis

The experimental data were expressed as mean ± SEM. The data were analyzed using ANOVA and Dunett's test. The results were considered statistically significance if $P < 0.05$.

RESULTS

Preliminary phytochemical analysis

The preliminary phytochemical analysis indicates the presence of flavonoids, terpenoids, saponins, alkaloids, tannins, steroids, deoxy sugars and gums, in the ethanol extract.

Table 1. Effect of *Mimosa pudica* on Carbon tetrachloride induced hepatotoxicity in rat's enzymes

Parameters	Group I (control)	Group II (Carbon tetra chloride group)	Group III (Test extract 200mg/kg	Group IV (Standard 100mg/kg))
SGOT (IU/L)	54.83±4.48	130.67±9.39a,**	116.5±6.42	53.83±4.05b,**
SGPT (IU/L)	70.5 ± 2.89	169.83±8.99a,**	116.5±7.94b,**	75.67±3.37b,**
ALP(IU/L)	54.17± .87	95.34± 7.12 a,**	73.17± 3.57 b,*	58.16±4.18b,**
Bilirubin(mg/dl)	0.75±0.065	2.68 ± 0.43 a,**	1.15± 0.27 b,**	0.93± 0.14 b,**
Total Protein(g/dl)	7.59 ± 0.25	4.98 ± 0.39 a,**	6.37 ± 0.31	7.2 ± 0.46 b,**

Values are expressed as mean ± S.E.M., n=6 in each group.
a, ** P < 0.001 as compared to normal control group,
b, ** P < 0.001, b,* P < 0.05, when compared with CCl4- treated control group.

Table 2. Effect of ethanol extract of *Mimosa pudica* on hepatic lipid peroxidation and antioxidant enzymes levels in CCl4- intoxicated rats.

Parameters	Group I (control)	Group II (Carbon tetrachloride group)	Group III (Standard 100mg/kg)	Group IV (Test extract 200mg/kg)
LPO(nM of MDA/mg of wet liver tissue)	2.4 ± 0.15	9.28±0.16**	2.8±0.27 b,**	5.8±0.34 b,**
GSH(μM/g of wet liver tissue)	21.42±0.55	7.33 ± 0.25**	19.02±1.02b,**	15.28±0.51b,**
SOD(IU/mg of wet liver tissue)	39.25±5.05	18.67± .48**	36.5± 3.07 b,**	28.83± 2.77 b,*
CAT(IU/ of wet liver tissue)	171.83±6.49	102.33±7.64**	167.5± 7.6 b,**	136.50±5.99b,**

Values are expressed as mean ± S.E.M., n=6 in each group.
a,** P < 0.001 as compared to normal control group,
b,** P < 0.001, b,** P < 0.05, when compared with CCl4- treated control group.

Acute oral toxicity study

Ethanolic extract of *Mimosa pudica* did not produce any toxic symptoms or mortality up to the dose level of 2000mg/kg body weight in rats, and hence the extract was considered to be safe and non-toxic for further pharmacological screening. The ethanol extract of *Mimosa pudica* (Mimosaceae) did not produced any mortality up to 3200 mg/kg b.w. Further dosing was not performed to estimate the LD50 (lethal dose) value. According to the OECD guidelines for the acute toxicity, anLD50 dose of 2000 mg/kg and the above is categorized as unclassified and hence the drug is found to be safe.

Hepatoprotective activity

The results of hepatoprtective action of *Mimosa pudica* on CCl4-treated rats with reference to biochemical changes in serum are shown in table 1. The CCl4-treated treated control group showed a significant (P < 0.001) increase in serum SGOT (130.67 ±9.39), SGPT (169.83 ± 8.99), ALP (95.34 ± 7.12) and total bilirubin (2.68 ± 0.43)levels and a decrease in total protein (4.98 ± 0.39), whereas serum SGOT, SGPT,ALP, bilirubin and total protein values showed 54.83 U/L 70.50 U/L, 54.17 ±4.87 U/L, 0.75 ± 0.06 mg/dl and 7.59 ± 0.25 mg/dl in normal control group, respectively. In contrast, animals treated with *Mimosa pudica* exhibited a significant decrease in SGOT (116.50 ±

6.42, SGPT (116.50 ± 7.94, ALP (73.17 ± 3.57) and total bilirubin (1.15 ± 0.27) along with a significant increase in total protein (6.37 ± 031).The standard drug silymarin (50 mg/kg) reduced the levels of SGOT (53.83 ±4.05), SGPT (75.67 ± 3.37), AST (58.16 ± 4.18) and total bilirubin (0.93 ± 0.14)along with a significant increase in total protein (7.20 ± 0.46) levels.

Antioxidant activity

The effects of *Mimosa pudica* at two dose levels (200 mg/kg b.w.) on enzymatic and non-enzymatic hepatic antioxidants and lipid peroxidation are shown in table2. In the CCl4-treated control rats, hepatic malondialdehyde (MDA) content was increased (9.28 ± 0.16), whereas this value showed (2.4 ± 0.15) in normal control. In contrast, hepatic MDA level in the groups treated with 200 mg/kg of *Mimosa pudica* decreased (5.8 ± 0.34 and 3.77 ± 0.57, respectively) significantly ($P < 0.001$) in a dose dependent manner towards normalization. There was a significant ($P < 0.001$) reduction in the activities of SOD (18.67 ± 1.48), CAT (102.33 ± 7.64), and GSH levels (7.33 ± 0.25) in liver, in CCl4-treated treated control rats as compared to the normal control rats. On the other hand, the hepatic SOD (28.83 ± 2.77), CAT (136.50 ± 5.99) activities, and GSH (15.28 ± 0.51) levels were significantly ($p < 0.001$) elevated in rats treated with *Mimosa pudica* (200 mg/kg b.w.), when compared with the CCl4 control rats.

DISCUSSION

The present studies were performed to assess the hepatoprotective and antioxidant activities of *Mimosa pudica* in rats against CCl4 as hepatotoxin, to prove its claims in folklore practice against liver disorders. The mechanism of hepatic damage byCCl4 is well documented. CCl4 is metabolized by cytochrome P450 enzyme system to tricholoromethyl free radical ($CCl3^{\bullet}$). This then readily interacts with molecular oxygen to form the trichloromethyl peroxy radical. Both tricholoromethyl and its peroxy radicals are capable of binding to proteins or lipids, or of abstracting a hydrogen atom from an unsaturated lipid, initiating lipidperoxidation and liver damage (Promod kumar *et al.,* 2008). The extent of hepatic damage is assessed by histological evaluation, enzymatic and non enzymatic antioxidant levels and lipid peroxide levels in liver, and the level of various biochemical parameters in circulation.

Assessment of liver function can be made by estimating the activities of serum GOT, GPT and ALP, which are originally present in higher concentration in the cytoplasm. Hepato cellular necrosis leads to the elevation of these serum marker enzymes, which are released from the liver into the blood stream (Ashok, 2002). Serum bilirubin and the total protein levels on the other hand are related to the functions of hepatic cells (Drotman and lawhorn, 1978). The increased levels of SGOT, SGPT, SALP and serum bilirubin are conventional indicator of liver injury (Achliya *et al.,*2004). The present study revealed a significant increase in the activities of SGOT, SGPT, SALP and serum bilirubin levels on exposure to CCl4, indicating considerable hepatocellur injury. Administration of *Mimosa pudica* at the dose of 200 mg/kg b.w significantly decreased the serum GOT, GPT, and ALP towards normal level, indicating the *Mimosa pudica* preserved the liver cell damage caused by CCl4, which is confirmed by enzymatic studies.

Abnormalities in serum total proteins and bilirubin are very common in hepatic toxicity. The rise in the levels of serum bilirubin is the most sensitive and confirms the intensity of jaundice (Drotman and lawhorn, 1978). It was found that the extract decreased the CCl4-induced elevated levels of bilirubin in groups III and IV, indicating the retrieval of structural integrity of the hepatocyte membranes or regeneration of damaged liver cells by the extracts. In this present study it was noted that the administration of CCl4 decreased the levels of total proteins. This parameter was brought back to the normal level in the group III and IV *Mimosa pudica* treated animals. The site-specific oxidative damage in some susceptible amino acids of proteins is now regarded as the major cause of metabolic dysfunction during pathogenesis (Uday *et al.,*1999). Attainment of near normaling rats confirms the hepatoprotective effect of the plant extract. A significant reduction ($P > 0.001$) was observed in SGPT, SGOT, ALP, and the total

bilirubin levels in the groups treated with silymarin. The enzyme levels were almost restored to the normal. The present study showed that the *Mimosa pudica* possesses hepatoprotective activity, as evidenced by the significant inhibition in the elevated levels of serum enzyme activities induced by CCl4. *Mimosa pudica* given orally (200 mg/kg b.w.)Once daily for 14 days showed dose-dependant hepatoprotective activity. The mechanism, by which *Mimosa pudica* exerts its protective action against CCl4- induced alternation in the liver, may be attributed to the antioxidant effect of the plant extract.

Antioxidant activities

High lipid peroxidation values indicate excessive free radical induced peroxidation. The measurement of lipid peroxide is also a marker of hepatocellular damage (Mohan *et al.,*2007). In animals treated with ethanolic extract and silymarin, the rise in lipid peroxides in liver tissue homogenate was prevented significantly. The decrease in lipid peroxides may be due to the antioxidant effect of the extract. Increase in MDA content by CCl4 oxidative damage, suggests enhanced lipid peroxidation leading to liver damage and failure of antioxidant defense mechanisms. Lipid peroxides are not only noxious to living organism, but also some of their stable breakdown products such as MDA has been recognized to cause some cell alterations by modifying protein structures. It has been shown that MDA is mutagenic to human cells (Niedernhofer *et al.,*2003) and play a significant role in DNA damage, sister-chromatid exchanges (SCEs) and carcinogenesis (Ray *et al.,*2001). The significant recduction in the MDA content in animal treated with *Mimosa pudica* suggests the production of liver through its inhibitory activity on lipid peroxidation.

GSH, being the most important intracellular reductant against chemically induced toxicity, can participate in the elimination of reactive intermediates by the reduction of hydroperoxides in the presence of GPx. GSH also functions as a free radical scavenger and aids the repair of radical caused biological damage(Meister, 1984). Deficiency of GSH within living organisms can lead to tissue disorder and injury. In the present study, a decrease in the levels of GSH in liver during hepatic injury was observed. The decrease in GSH level represents increased utilization due to oxidative stress (Anuradha and Selvam, 1993).Administration of *Mimosa pudica* (200 mg/kg b.w, p.o) increased the content of GSH in liver of CCl4- intoxicated rats. Reduced activities of SOD and CAT in liver have been observed during CCl4 liver injury. SOD is an important antioxidant enzyme which catalyses the dismutation of superoxide radicals (McCord *et al.,*1976). CAT is a heme prote in which catalyses the reduction of hydrogen peroxides and protects the tissue from highly reactive hydroxyl free radicals (Chance *et al.,*1982). Therefore, the reduction in the activity of these enzymes (SOD, CAT) may result in oxidative tissue damage due to the accumulation of superoxide radicals and hydrogenperoxide. In the present study**,** it was observed that the *Mimosa pudica* caused a significant increase in the hepatic SOD and CAT activities of the CCl4 intoxicated rats. This means that the *Mimosa pudica* can reduce reactive oxygen free radicals and improve the activities of the hepatic antioxidant enzymes. *Mimosa pudica* treated rats proved that the hepatic damage might be protected by their potent antioxidant property. Wide verities of naturally occurring compounds can protect the liver and other tissue from damage due to its antioxidant properties. Flavonoids are known to possess hepatoprotective activity by scavenging the free radicals and elevating the liver antioxidatant enzymes (Manjunatha, 2008; Baek *et al.,*1996). Presence of flavanoids in *Mimosa pudica* may be responsible for the protective effect on CCl4-induced liver damage in rats. Further research is going on to pin point the exact molecular mechanism of *Mimosa pudica* involved for liver protection against various models.

CONCLUSION

In accordance with these results, it may be confirmed due to the presence of phytoconstituents such as flavonoids, alkaloids and glycosides which are present in the ethanolic extract could be considered as, responsible for the significant hepatoprotective activity. In conclusion, it can be said that the ethanolic extract of *Mimosa pudica*

exhibited a hepato protective effect against Carbon tetrachloride induced hepatotoxicity. Further investigation is underway to identify the exact phyto constituents which are responsible for its hepatoprotective effect.

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