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Hepatoprotective activity of *Carallia brachiata*

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Abstract: Ethyl acetate and methanol extracts of the *Carallia brachiata* bark were tested for hepatoprotective activity against carbon tetrachloride (CCl_4) induced hepatotoxicity in rats at dose levels of 250 and 400 mg/kg body weight. The ethyl acetate extract has shown high significance by lowering the biochemical parameters such as serum glutamate pyruvate transaminase (SGPT), serum glutamate oxaloacetate transaminase(SGOT), alkaline phosphatase (ALP) and serum bilirubin when compared with the hepatic control and almost similar to that of standard silyramin. The methanol extract has shown less significant activity when compared with that of ethyl acetate extract.

Key words: *Carallia brachiata,* hepatoprotective, carbon tetrachloride.

Introduction

Liver is one of the largest organs in human body and the major site for metabolism and excretion. It is involved with almost all the biochemical pathways of growth, fight against disease, nutrient supply, energy provision and reproduction [1]. Jaundice and hepatitis are two major liver disorders that account for a high death rate. Modern medicines have little to offer for alleviation of hepatic diseases and it is chiefly the plant based preparations are employed for the treatment of many liver disorders[2]. Therefore, many folk remedies from plant origin are to be tested for their potential hepatoprotective and antioxidant properties in liver damage in experimental animal models. Carbon tetrachloride (CCl_4) induced hepatotoxicity model is widely used for the study of hepatoprotective effects of drugs and plant extracts [3, 4].

Carallia brachiata (F: Rhizophoraceae) is commonly known as karalla, is a large evergreen ornamental tree. It is traditionally used for treating itch, oral ulcer, inflammation of throat and stomatitis[5]. The ethyl acetate and methanol extracts of bark exhibited anti-inflammatory[6], wound healing [7] and antimicrobial activities[8]. From bark, proanthocyanidins namely carallidin, mahuannin A and para-hydroxy benzoic acid were reported[9]. However no reports are available on the efficacy of the *C. brachiata* bark as hepatoprotective agent. Therefore an attempt has been made to investigate the hepatoprotective activity of stem bark of *C. brachiata* against carbon tetrachloride (CCl_4) induced hepatotoxicity in rats.

Materials and methods

Plant material and Preparation of Extracts

Carallia brachiata stems were collected from Tirupathi forest ranges, A.P, India in September 2006.The plant was identified and authenticated by Prof. K Madhav Chetty, Department of Botany, S V University,Tirupathi, India. A voucher specimen (CB-10-06) is maintained in phytochemistry and pharmacognosy, department of G.Pulla Reddy College of pharmacy, Hyderabad, A.P,India.

The bark was separated from stems, air dried and grounded to coarse powder and extracted successively with pet ether, ethyl acetate and methanol by cold maceration. All the extracts were concentrated and

dried in a dessicator. Qualitative phytochemical tests were performed for phytoconstituents.

Experimental animals

Male Wistar rats (150-200g) were used to carryout the hepatoprotective activity. They were maintained under standard environmental conditions and had free access to feed (Nutrient animal feed, Rayan Biotechnology Pvt. Ltd) and water *ad libitium* during quarantine period. The institutional animal ethics committee of G. Pulla Reddy College of Pharmacy, Hyderabad, A.P, India approved the animal experimental protocol.

Hepatoprotective effect against Carbon tetrachloride induced hepatotoxicity in rats

The animals were divided randomly into seven groups of six rats each. The hepatoprotective activity of the plant extracts was tested using CCl_4 model. Group I (normal control) received 2% gum acacia suspension orally for seven days. Group II Hepatic control. Group III IV received ethyl acetate extract of *C. brachiata* at an oral dose of 250 and 400 mg/kg respectively. Group V – VI received methanol extract of *C. brachiata* at an oral dose of 250 and 400 mg/kg respectively. Group VII served as standard and received Silyramin at a dose of100 mg/kg orally for 7 days. On 7th day Group II-VII, 30 min post dose of extract administration animals received carbon tetrachloride at the dose of 1 ml/kg (1:1 of carbon tetrachloride in liquid paraffin) orally [10,11]. After 24 hrs of administration of acute dose of CCl_4 blood from each rat was withdrawn by retro-orbital puncture and collected in previously labeled centrifuging tubes and allowed to clot for 30 min at room temperature. Serum was separated by centrifugation at 3000 rpm for 15 minutes. The separated serum was used for the estimation of biochemical parameters like serum glutamate pyruvate transaminase (SGPT), serum glutamate oxaloacetate transaminase (SGOT) [12], and alkaline phosphatase (ALP) [13] and serum bilirubin.

Statistical Analysis

The results are expressed as the mean± SEM. Statistical differences were evaluated using a one-way analysis of variance (ANOVA) followed by Dunnett´s test. Results were considered to be statistically significant at $p<0.001$

Table: Effect of *C. brachiata* extracts on biochemical parameters in carbon tetrachloride induced hepatic injury in rats

Groups	Treatment	SGPT IU/L (Mean ± SEM)	SGOT IU/L (Mean ± SEM)	ALP IU/L (Mean ± SEM)	Bilirubin IU/L (Mean ± SEM)
Group I	Control	30.3 ± 1.21	25.06 ± 1.96	73.67 ± 1.03	4.58 ± 0.86
Group II	Hepatic Control	162 ± 0.89	38 ± 1.41	151 ± 2	6.77 ± 0.99
Group III	Ethyl Acetate Extract 250 mg/kg	54.8 ± 1.47**	29.68 ± 1.79**	151.3 ± 1.75	4.5 ± 1.4*
Group VI	Ethyl Acetate Extract 400 mg/kg	42 ± 2.28**	23.9 ± 1.74**	62.83 ± 2.25**	2.5 ± 0.7**
Group V	Methanol Extract 250 mg/kg	149 ± 2.8**	49.96 ± 1.92**	148.45 ± 2.19**	6.16 ± 2.85
Group VI	Methanol Extract 400 mg/kg	98 ± 1.41**	35.24 ± 1.71	121 ± 1.79**	5 ± 0.77
Group VII	Silymarin	35.8 ± 1.72**	22.57 ± 2.27**	61.53 ± 1.63**	3.58 ± 1.24**

Values are (Mean ± SEM); n=6, *$p<0.001$ vs Group II

Results

The results of hepatoprotective activity of ethyl acetate and ethanol extracts of *C.brachiata* on rats intoxicated with CCl_4 were illustrated in table. The results are expressed as mean ± SEM. The animals treated with CCl_4cause significant increase in serum SGPT, SGOT, ALP and total bilirubin compared to normal rats, indicating hepatotoxicity. In contrast pre treatment with ethyl acetate extract (250 and 400 mg/kg p.o) showed significant ($P<0.001$) reduction where as ethanol extract (250 and 400 mg/kg p.o) showed less significant reduction in serum enzymes in a dose dependent manner compared to toxic control. The hepatoprotective activity exhibited by ethyl acetate extract at a dose of 400mg/kg was comparable to that of standard sylmarin.

Discussion

The liver can be injured by many chemicals and drugs. Liver damage induced by CCl_4 is commonly used model for the screening of hepatoprotective drugs. The CCl_4 is converted into reactive metabolite, halogenated free radical by hepatic cytochrome P450s[14] which in turn covalently binds to cell membrane and organelles to elicit lipid peroxidation with subsequent tissue injury.

The qualitative phytochemical investigation of extracts *of Carallia brachiata* showed positive test for carbohydrates, triterpenes, steroids, fats and fixed oils, tannins, phenols and flavonoids.

The results of biochemical parameters revealed the elevation of enzyme level in CCl_4-treated group, indicating that CCl_4 induces damage to the liver. A significant reduction ($P<0.001$) was observed in SGPT, SGOT, ALP and total bilirubin levels in the groups treated with ethyl acetate extract of *C. brachiata*.

Conclusion

In conclusion, decrease in biochemical parameters in hepatic damaged rats treated with extracts indicates the effectiveness of *C. brachiata* as hepatoprotective. The preliminary phytochemical studies revealed the presence of flavonoids in ethyl acetate and methanol extracts of *C.brachiata*. Since flavonoids have been reported for their hepatoprotective activity[15], it may be speculated that these constituents of *Carallia* are responsible for the observed protective effects.

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