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 (CCl4) 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 reproduction1. 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 disorders2. 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 (CCl4) induced hepatotoxicity model is widely used for the study of hepatoprotective effects of drugs and plant extracts3,4. 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 stomatitis5. The ethyl acetate and methanol extracts of bark exhibited anti-inflammatory6, wound healing7 and antimicrobial activities8. From bark, proanthocyanidins namely carallidin, mahuannin A and para-hydroxy benzoic acid were reported9. 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 (CCl4) 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 libitum 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 CCl₄ 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 of 100 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. After 24 hrs of administration of acute dose of CCl₄ 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), and alkaline phosphatase (ALP) 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>
<thead>
<tr>
<th>Groups</th>
<th>Treatment</th>
<th>SGPT IU/L (Mean ± SEM)</th>
<th>SGOT IU/L (Mean ± SEM)</th>
<th>ALP IU/L (Mean ± SEM)</th>
<th>Bilirubin IU/L (Mean ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>Control</td>
<td>30.3 ± 1.21</td>
<td>25.06 ± 1.96</td>
<td>73.67 ± 1.03</td>
<td>4.58 ± 0.86</td>
</tr>
<tr>
<td>Group II</td>
<td>Hepatic Control</td>
<td>162 ± 0.89</td>
<td>38 ± 1.41</td>
<td>151 ± 2</td>
<td>6.77 ± 0.99</td>
</tr>
<tr>
<td>Group III</td>
<td>Ethyl Acetate Extract 250 mg/kg</td>
<td>54.8 ± 1.47**</td>
<td>29.68 ± 1.79**</td>
<td>151.3 ± 1.75</td>
<td>4.5 ± 1.4*</td>
</tr>
<tr>
<td>Group VI</td>
<td>Ethyl Acetate Extract 400 mg/kg</td>
<td>42 ± 2.28**</td>
<td>23.9 ± 1.74**</td>
<td>62.83 ± 2.25**</td>
<td>2.5 ± 0.7**</td>
</tr>
<tr>
<td>Group V</td>
<td>Methanol Extract 250 mg/kg</td>
<td>149 ± 2.8**</td>
<td>49.96 ± 1.92**</td>
<td>148.45 ± 2.19**</td>
<td>6.16 ± 2.85</td>
</tr>
<tr>
<td>Group VI</td>
<td>Methanol Extract 400 mg/kg</td>
<td>98 ± 1.41**</td>
<td>35.24 ± 1.71</td>
<td>121 ± 1.79**</td>
<td>5 ± 0.77</td>
</tr>
<tr>
<td>Group VII</td>
<td>Silymarin</td>
<td>35.8 ± 1.72**</td>
<td>22.57 ± 2.27**</td>
<td>61.53 ± 1.63**</td>
<td>3.58 ± 1.24**</td>
</tr>
</tbody>
</table>

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<sub>4</sub> were illustrated in table. The results are expressed as mean ± SEM. The animals treated with CCl<sub>4</sub> cause 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 whereas 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<sub>4</sub> is commonly used model for the screening of hepatoprotective drugs. The CCl<sub>4</sub> is converted into reactive metabolite, halogenated free radical by hepatic cytochrome P450<sub>14</sub> which in turn covalently binds to cell membrane and organelles to elicit lipid peroxidation with subsequent tissue injury.

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, it may be speculated that these constituents of *Carallia* are responsible for the observed protective effects.

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References


