INTRODUCTION
Liver is one of the most important organs in the biotransformation of food, drugs, and endogenous and exogenous substances. Herbal plants have been recently popularized in modern medicine, since many therapeutically important compounds are derived from them. From the literature review it is observed that many species belonging to the Tulasi family or Lamiaceae are proven hepatoprotective agents. Hence, the present study was conducted to evaluate the hepatoprotective potentials of Leonotis nepaetifolia (L.)R.Br., a common Lamiaceous member in and around Trichy, India. Medicinal plants have been recently popularized in modern medicine, since many therapeutically important compounds are derived from them. The extracts of these plants are commonly used in the treatment of liver diseases like hepatitis, cirrhosis etc. [5]. Hence, the present study focuses on the hepatoprotective activity of Leonotis nepaetifolia (L.)R.Br. in CCl4 induced toxicity in Albino rats.

MATERIALS AND METHODS

Collection of plant material
The whole plant Leonotis nepaetifolia (L.)R.Br. was collected in November from and around Trichy, identified with the help of Floras of Presidency of Madras and authenticated with the voucher specimen deposited at the Rabinet herbarium of St. Joseph’s College, Trichy.

Preparation of aqueous extract
The authenticated aerial parts of the plant were shade dried and coarsely powdered. The powder was mixed thoroughly with 6 times the volume of water and stirred continuously for a period of 24 hours. The extract was filtered with muslin cloth. The residue was re extracted. The filtrate was mixed and evaporated in a water bath till it reached a thick consistency. The extract was stored in refrigerator till further use.

Experimental models
Wistar strains of Albino rats of both sexes weighing 150-200g were used for the study. Animals were housed in well ventilated cages in the CPCSEA approved animal house. Animals were divided into six groups consisting of six rats each. Group 1 served as normal control. Group 2 served as the disease control. The rats received 0.5ml CCl4/150g bd wt for a period of 21 days. Group 6 was induced with CCl4 and treated with silimarin at a dose of 25mg/kg bd wt. The effects of the plant extract and silimarin on serum transaminases (AST & ALT), alkaline phosphatase (ALP), total protein and bilirubin were measured in the hepatotoxic rats induced by CCl4. Further, the effects of the plant extract on Lipid peroxidation (LPO), enzymatic antioxidants – superoxide dismutase (SOD), glutathione peroxidase (GPx), Catalase (CAT) and non-enzymatic antioxidant – reduced glutathione (GSH) and Lipid peroxidation (LPO) were studied.

RESULTS

The animals were sacrificed at the end of the experimental period the animals were sacrificed by cervical decapitation. The blood and liver tissue was collected and used for the studies.

The data of results obtained were subjected to statistical analysis and expressed as mean ± SD. The data were statistically analyzed by one-way analysis of variance (ANOVA) and p<0.05 was considered to be significant.

Table 1. Effect of Leonotis nepaetifolia (L.)R.Br. on the biochemical parameters and serum hepatic marker enzymes in CCl4 induced toxicity

<table>
<thead>
<tr>
<th>Groups</th>
<th>Bilirubin (mg/dl)</th>
<th>Total Protein (mg/dl)</th>
<th>SGOT (U/L)</th>
<th>SGPT (U/L)</th>
<th>ALP (U/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GpI Normal control</td>
<td>0.98 ± 0.045</td>
<td>7.2 ± 0.03</td>
<td>7.34 ± 0.7</td>
<td>41.7 ± 0.66</td>
<td>597.2 ± 0.65</td>
</tr>
<tr>
<td>GpII Disease control</td>
<td>5.46 ± 0.15</td>
<td>2.5 ± 0.02</td>
<td>145 ± 1.67</td>
<td>215 ± 0.65</td>
<td>1003 ± 0.88</td>
</tr>
<tr>
<td>GpIII (100mg/kg body wt)</td>
<td>3.6 ± 0.43</td>
<td>4.56 ± 0.37</td>
<td>103.17 ± 6.5</td>
<td>47.8 ± 4.5</td>
<td>278.8 ± 5.0</td>
</tr>
<tr>
<td>GpIV (200mg/kg body wt)</td>
<td>4.56 ± 0.19</td>
<td>5.61 ± 0.24</td>
<td>64.3 ± 7.5</td>
<td>206 ± 4.7</td>
<td>113.2 ± 6.93</td>
</tr>
<tr>
<td>GpV (300mg/kg body wt)</td>
<td>0.87 ± 0.074</td>
<td>0.03 ± 0.137</td>
<td>6.7 ± 1.8*</td>
<td>8.7 ± 1.25</td>
<td>24.3 ± 2.44*</td>
</tr>
<tr>
<td>GpVI (Silymarin 25mg/kg body wt)</td>
<td>1.01 ± 0.06</td>
<td>6.90 ± 0.127</td>
<td>7.67 ± 0.27*</td>
<td>11.93 ± 0.49</td>
<td>555.8 ± 4.51*</td>
</tr>
</tbody>
</table>

Values are ± SEM; n=6
*p<0.05 statistically significant when compared with normal control
#p<0.05 statistically significant when compared with CCl4 treated group

SGOT - Serum Glutamiate Oxaloacetate Transaminase
SGPT - Serum Glutamiate Pyruvate Transaminase
ALP - Alkaline Phosphatase
Table II. Antioxidant status of the models treated with Leonotis nepetifolia (L.) R. Br. in CCl₄ induced toxicity

<table>
<thead>
<tr>
<th>Groups</th>
<th>LPO (nmol/mg of protein)</th>
<th>SOD (U/mg of protein)</th>
<th>GSH (nmol/mg)</th>
<th>GPx (nmol/mg)</th>
<th>CAT (nmol/mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gpl Normal control</td>
<td>2.53 ± 0.12</td>
<td>3.01 ± 0.02</td>
<td>44.4 ± 0.31</td>
<td>45 ± 0.35</td>
<td>64.4 ± 0.31</td>
</tr>
<tr>
<td>GPII Disease control (CCl₄)</td>
<td>5.9 ± 0.14</td>
<td>0.78 ± 0.04</td>
<td>31 ± 0.365</td>
<td>27 ± 0.33</td>
<td>52.3 ± 0.35</td>
</tr>
<tr>
<td>GpiIV (100mg/kg body wt)</td>
<td>4.0 ± 0.11†</td>
<td>1.1 ± 0.07†</td>
<td>39 ± 0.28²</td>
<td>29.6 ± 0.26</td>
<td>58.8 ± 0.36²</td>
</tr>
<tr>
<td>GpiV (200mg/kg body wt)</td>
<td>3.72 ± 0.12*</td>
<td>1.55 ± 0.033</td>
<td>48.0 ± 0.29³</td>
<td>39.5 ± 0.28²</td>
<td>62.9 ± 0.53³</td>
</tr>
<tr>
<td>GpiVI (Silymarin 25mg/kg body wt)</td>
<td>2.98 ± 0.12²</td>
<td>2.6 ± 0.02</td>
<td>42.0 ± 0.23³</td>
<td>41.6 ± 0.31</td>
<td>42.4 ± 0.13°</td>
</tr>
</tbody>
</table>

Values are ± SEM, n=6

*p < 0.05 statistically significant when compared with disease control group

**p < 0.05 statistically significant when compared with normal control group

The antioxidant enzymes SOD, CAT, GPx and non-enzymatic antioxidant GSH were reduced which mediated the lipid per oxidation of the membranes resulting in an elevation of LPO in group II animals. The treatment with the test drug restored the antioxidant status which in turn served to scavenge the formed free radicals. (Table II)

DISCUSSION

The changes associated with CCl₄ induced liver damage are similar to that of acute viral hepatitis. CCl₄ is bifunctional to trichloromethyl free radical which binds covalently to the cell membrane and organelles to elicit lipid per oxidation and finally cell death[15]. The administration of CCl₄ increases the level of the serum marker enzymes AST, ALT, ALP and GGT indicating the hepatic damage. The serum enzyme levels are powerful tools in the identification of hepatotoxicity[15]. CCl₄ mediated toxicity increases the permeability of the membranes and results in cellular leakage[16]. Restoration of the levels of marker enzymes in the serum indicated the tissue regeneration property of the aqueous extract of Leonotis nepetifolia (L.) R. Br.

The stress induced was nullified by the extract of Leonotis nepetifolia (L.) R. Br. which was observed from the restoration of the SOD and catalase activity.

CONCLUSION

From the Data of results obtained it is evaluated that the common weed Leonotis nepetifolia (L.) R. Br. possesses a significant hepatoprotective potential in a dose dependent manner. The study also helped us to identify the therapeutic values of the common plants present around us.

REFERENCES


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