Antiulcer Activity of Patol Churna against Experimental Gastro-duodenal Ulcers in Rats
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Abstract
Patol churna is a well known ayurvedic formulation of *Trichosanthes cucumerina* Linn. (*cucurbitaceae*) administered in case of number of alimentary and liver disorders. It is widely used in Indian folk medicine for variety of disease conditions. The aim of present study was to evaluate the antilulcer activity of 50% ethanolic extract of patol churna (PCE) using various experimental models of gastric and duodenal ulceration in rats. Oral administration of 50% ethanolic extract of patol churna was evaluated in rats against ethanol, aspirin and pylorus ligated gastric ulcers as well as cysteamine-induced duodenal ulcers. In all the models studied, the antilulcer activity of PCE compared with that of cimetidine (100mg/kg, p.o.), an H<sub>2</sub> receptor antagonist. PCE showed significant antilulcer activity in ethanol-induced and aspirin-induced gastric ulcer models. In 19 hrs pylorus ligated rats, significant reduction in ulcer index, total acidity and pepsin activity was observed with PCE, when compared with the control group. Mucosal defensive factors such as pH, mucin activity and gastric wall mucous content was found to be increased with PCE. PCE was also, afforded remarkable protection in cysteamine-induced duodenal lesions. The antilulcer activity of PCE was comparable with that of cimetidine. Thus, patol churna extract possess significant antilulcer activity against both gastric and duodenal ulcers in rats. The antilulcer activity may be attributed to its cytoprotective action and inhibition of acid secretory parameters.

Keywords: Antilulcer activity; Duodenal ulcer; Gastric ulcer; *Trichosanthes cucumerina* Linn; Patol churna

Introduction
*Trichosanthes cucumerina* Linn. (*cucurbitaceae*) is an annual climber and widely distributed throughout India, Ceylon, Malaya and North Australia. In Gujarat, the plant is known as 'Patola' or 'Kadvi Parval'. Patol churna is a well known ayurvedic formulation of *T. cucumerina* administered in case of number of alimentary and liver disorders. Whole plant is reputed for the treatment of hepatic and alimentary canal disorders. Fruits of *Trichosanthes cucumerina* are used as laxative, purgative, antipyretic, alexiteric and antilulcer agent. The leaves are good for bilious disorders [1]. Antidiabetic [2], hepatoprotective [3], anti-inflammatory [4], antifertility [5], antioxidant [6], antibacterial [7], antifungal [8] and antiviral [9] activities of the plant were reported. The fruits contain ascorbic acid, lycopene, phenols, flavonoids, alkaloids, tannins and saponins [6,10]. Present study was undertaken to evaluate the effect of 50% ethanolic extract of patol churna (PCE) in various experimental ulcer models.

Materials and Methods

Plant material and extraction
Patol churna, a readymade formulation powder was procured from the local market of Ahmedabad, India and authentication was done in the department of Pharmacognosy, L. M. College of Pharmacy, Ahmedabad. It was found to be a mixture of all the aerial parts of *Trichosanthes cucumerina*. The powder was extracted exhaustively with 50% ethanol by maceration at room temperature for 2 days with occasional shaking. The crude extract was dried at 40°C under vacuum. Freshly prepared aqueous solution of dried extract of patol churna (PCE) in suitable dilution was administered orally in the test animals. For the ethanol induced ulcer model, animals were divided in to five groups, each group consisting of six animals. Group 1 served as control group received distilled water (vehicle) 1 ml/kg, p.o., group 2-4 served as test groups received PCE (300, 500 and 800mg/kg, p.o.) and group 5 served as positive control group received cimetidine (100mg/kg, p.o.).

Drugs and chemicals
Cimetidine (Cadila, Ahmedabad) was used as reference standard. Aspirin (Cadila, Ahmedabad) and Cysteamine (Merck, Germany) were used for experimental induction of gastric and duodenal ulcers respectively.

Animals
Wister rats (200-250g) of either sex bred in Central Animal House facility of the institute were used. The animals were housed under standard conditions, maintained on a 12 hrs light/dark cycle and had free access to food and water up to the time of experimentation. The animals were acclimatized to the laboratory environment 1 hr before the experiments. Animals were randomly distributed into groups of 10 animals each. All experiments were conducted during the light period (08.00-16.00 hrs). All the protocols were approved by the Institutional Animal Ethical Committee (IAEC) and conducted according to the guidelines of CPCSEA (Committee for the Purpose of Control and Supervision of Experiment on Animals).

Treatment
Freshly prepared aqueous solution of dried extract of patol churna (PCE) in suitable dilution was administered orally in the test animals. For the ethanol induced ulcer model, animals were divided in to five groups, each group consisting of six animals. Group 1 served as control group received distilled water (vehicle) 1 ml/kg, p.o., group 2-4 served as test groups received PCE (300, 500 and 800mg/kg, p.o.) and group 5 served as positive control group received cimetidine (100mg/kg, p.o.).

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Positive control, cimetidine treated animals also showed significant reduction in ulcer index as compared to control animals (p< 0.05).

**Pylorus ligation-induced gastric ulcer model**

As shown in Table 3, PCE and cimetidine showed significant reduction in ulcer index (p< 0.05) as compared to control. None of the treatment groups showed any marked change in volume of gastric acid secretion parameter. There was significant rise in gastric pH by PCE and cimetidine as compared to control group (Table 3). The treatment groups viz. PCE and cimetidine showed significant reduction in total acidity when compared with the control group (Table 3). Total acid output remained unaltered in all the treatment groups. Along with total acidity, pepsin activity was significantly reduced by PCE and cimetidine treatment (Table 3). Significant rise in total carbohydrate content was observed in treatment groups as compared with the control.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg, p.o.)</th>
<th>Ulcer Index</th>
<th>% Protection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>-</td>
<td>2.19 ± 0.36</td>
<td>-</td>
</tr>
<tr>
<td>PCE</td>
<td>300</td>
<td>0.91 ± 0.14*</td>
<td>58.45</td>
</tr>
<tr>
<td></td>
<td>500</td>
<td>0.59 ± 0.08*</td>
<td>73.06</td>
</tr>
<tr>
<td>Cimetidine</td>
<td>100</td>
<td>0.39 ± 0.08*</td>
<td>82.19</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>1.17 ± 0.08*</td>
<td>46.58</td>
</tr>
</tbody>
</table>

n = 6 Expressed as mean ± SEM. One way Anova followed by Tukey’s multiple range test; *p<0.05 when compared with control group.

**Table 1:** Effect of PCE against ethanol–induced gastric ulcer model in rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>PCE (500 mg/kg) (p.o.)</th>
<th>Cimetidine (100 mg/kg) (p.o.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcer index</td>
<td>0.66 ± 0.11</td>
<td>0.11 ± 0.03*</td>
<td>0.14 ± 0.05*</td>
</tr>
<tr>
<td>Vol. of gastric content (ml/100g)</td>
<td>3.67 ± 0.24</td>
<td>4.00 ± 0.56</td>
<td>4.15 ± 0.13</td>
</tr>
<tr>
<td>pH</td>
<td>2.20 ± 0.19</td>
<td>3.62 ± 0.19*</td>
<td>5.20 ± 0.07*</td>
</tr>
<tr>
<td>Total acidity (mEq/L)</td>
<td>14.77 ± 0.94</td>
<td>5.03 ± 0.33*</td>
<td>9.83 ± 0.20*</td>
</tr>
<tr>
<td>Total acid output (mEq/100 g)</td>
<td>54.04 ± 4.70</td>
<td>46.27 ± 15.3</td>
<td>40.22 ± 0.88</td>
</tr>
<tr>
<td>Pepsin activity (µg/ml)</td>
<td>750 ± 41.03</td>
<td>299.63 ± 17.62*</td>
<td>310.0 ± 31.97*</td>
</tr>
</tbody>
</table>

n = 6 Expressed as mean ± SEM. One way Anova followed by Tukey’s multiple range test; *p<0.05 when compared with control group.

**Table 2:** Effect of PCE against aspirin-induced gastric ulcer model in rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>PCE (500 mg/kg, p.o.)</th>
<th>Cimetidine (100 mg/kg, p.o.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total carbohydrate (µg/ml)</td>
<td>496.67 ± 46.69</td>
<td>1506.33 ± 100.16*</td>
<td>880.5 ± 54.45*</td>
</tr>
<tr>
<td>Protein content (µg/ml)</td>
<td>294.7 ± 67.04</td>
<td>129.5 ± 21.78*</td>
<td>46.17 ± 1.14*</td>
</tr>
<tr>
<td>TC : PR ratio</td>
<td>2.18 ± 0.41</td>
<td>13.64 ± 2.24*</td>
<td>19.08 ± 1.12*</td>
</tr>
<tr>
<td>GWMC</td>
<td>57.63 ± 7.90</td>
<td>59.18 ± 6.12</td>
<td>74.21 ± 7.99</td>
</tr>
</tbody>
</table>

n = 6 Expressed as mean ± SEM. One way Anova followed by Tukey’s multiple range test; *p<0.05 when compared with control group.

**Table 3:** Effect of PCE on ulcer index and acid secretory parameters in pylorus ligated gastric ulcers in rats.
cells in the fundic mucosa [24]. Also, certain prostaglandins are involved in gastric hypersecretion, involving the release of gastrin, histamine and somatostatin [25]. In gastric hypersecretion, the release of gastrin, histamine and somatostatin is significant. The pathogenesis of cysteamine-induced ulcers is considered to be due to continuous hypersecretion of gastric acid [29]. The pathogenesis of cysteamine-induced duodenal ulcers includes enhanced gastric acid secretion [29], increased duodenal motility [30], delayed gastric emptying [31] and decreased duodenal bicarbonate secretion in response to acid [31]. It is suggested from our results that patol churna and cimetidine possess significant antiduodenal ulcer activity. The mechanism of this activity can be related to inhibitory effect of acid and pepsin activity.

Cysteamine-induced duodenal ulcer model

In cysteamine-induced duodenal ulcer model, PCE and cimetidine showed significant reduction in the total lesion area when compared with control group (Table 5).

Summary of the present study indicate that 50% ethanolic extract of patol churna has protective effect against experimental gastro-duodenal ulcers in rats.

#### Table 5: Effect of PCE on cysteamine-induced duodenal ulcer in rats.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Ulcer incidence</th>
<th>Mortality</th>
<th>Ulcer score</th>
<th>Total lesion area (mm²)</th>
<th>Ulcer index</th>
<th>% inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>8/8 100</td>
<td>3/8 37.5</td>
<td>2.50 ± 0.28</td>
<td>88.38 ± 3.87</td>
<td>4.5</td>
<td>-</td>
</tr>
<tr>
<td>PCE (500 mg/kg, p.o.)</td>
<td>8/8 100</td>
<td>1/8 12.5</td>
<td>1.00 ± 0.12</td>
<td>43.75 ± 1.70*</td>
<td>3.0</td>
<td>33.33</td>
</tr>
<tr>
<td>Cimetidine (100mg/kg, p.o.)</td>
<td>8/8 100</td>
<td>0/0 0.0</td>
<td>0.90 ± 0.12*</td>
<td>41.2 ± 3.04*</td>
<td>2.9</td>
<td>35.56</td>
</tr>
</tbody>
</table>

n=6 Expressed as mean ± SEM. One way Anova followed by Tukey’s multiple range test; *p<0.05 when compared with control group.

**References**


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