EVALUATION OF ANTI ULCER ACTIVITY OF BETA VULGARIS IN RATS

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Abstract

The present study was designed to evaluate the anti ulcer effect of ethanolic extract of beet root containing flavonoids{(+)-Dehydrovomifoliol, 3-Hydroxy-5a,6a-epoxy-b-ionone, Vitexin 7-O-b-D-glucopyranoside, Vitexin 2'-O-b-D-glucopyranoside} and phenolic compounds on physical and chemical factors induced gastric ulcer models in rats. To assess the anti ulcer activity of EEBV, UI, % of ulcer protection were determined in aspirin and alcohol induced methods and UI, % of ulcer protection, Volume of gastric content, Volume of Gastric juice, PH, Total acidity and Free acidity were determined in pylorus ligation method. The Phytochemical screening of EEBV revealed the presence of Carbohydrates, Glycosides, Proteins & amino acids, Saponins, Tannins & Phenolic compounds and Flavonoids. The EEBV shows protection in dose dependent manner against characteristic lesions produced by ethanol and aspirin reduced values of ulcer index as compared control group. The Ethanolic extract of betavulgaris and ranitidine significantly decreased the total acidity and free acidity; and significantly enhance the PH. From these results it is concluded that the ethanolic extract of beta vulgaris at the doses of 250mg/kg and 400 mg/kg clearly demonstrated antiulcer property in experimental model of rats.

Key words: Peptic Ulcer, Beta Vulgaris, Pylorus Ligation, Ranitidine.

I. Introduction:

Peptic ulcer is one of the major gastro-intestinal disorders. Peptic ulcer is a lesion of gastric or duodenal mucosa, it occur due to an imbalance between the offensive (gastric acid secretion) and defensive (gastric mucosal integrity) factors. Most injurious agents such as acid, pepsin, bile acids, food ingredients, bacterial products and certain drugs and pathological condition such as Zollinger –Ellison Syndrome, they cause the ulcers in gastric or duodenal mucosa
The erosion on the stomach, it is referred to as a gastric ulcer. If it is in the duodenum (the part of the small intestine just after the stomach), it is called a duodenal ulcer.

Peptic ulcer disease is a worldwide problem, affecting about 1 in 10 people. In the early 20th century peptic ulcers were thought to be caused by emotional stress and spicy foods. Peptic ulcer is more frequent in men than in women. After 45 years of age peoples have less sex differences probably because the incidence of ulcer increases in post menopausal women. The ulcer differences between sexes are related in some way to sex hormones and that the female sex hormones protect against ulceration \[3\]. Proton pump inhibitors and H2- receptor antagonists are the most widely used drugs to treat peptic ulcer disease. They produce some adverse reactions such as hypersensitivity, arrhythmia, impotence and haemopoietic changes, gynaecomastia, alopecia, mental confusion with is a possibility of increased rate of ulcer recurrence within one year after cessation of the treatment \[4\]. Thus, there is a need for more effective, less toxic and cost-effective anti-ulcer agents. In recent years, a widespread search has been launched to identify new anti-ulcer drugs from natural herbal sources. Numerous medicinal plants and their formulations are used for peptic ulcer in ethno medical practices and in traditional system of medicine in India. However, we do not have satisfactory remedy for serious ulcer disorder; most of the herbal drugs speed up the natural ulcer healing process. One such anti ulcer activity possessing natural source is \textit{beta vulgaris} as per traditional system of medicine.

\textit{Beta vulgaris} (Linn.), belonging to family chenopodiaceae, is a native of South Europe \[5\]. Beetroots contain a unique class of water-soluble, nonphenolic antioxidants, the betalains, including two classes of compounds, red betacyanins (principally betanin) and yellow betaxanthines. Beets contain an abundance of minerals: potassium, phosphorus, calcium, sulphur, iodine, iron, manganese, chlorine, and copper, as well as traces of the rare metals rubidium and cesium. Heat labile phenolic compounds and flavonoids also present. The antioxidant effects of betalains have been demonstrated mainly in various in vitro experiments \[6\].

\textit{Beta vulgaris} some reports indicating the chemopreventive activity \[7\], hepatoprotective activity \[8\], anti inflammatory activity \[9\], wound healing \[10\], Anti oxidant activity \[11\], anti Depressant activity \[12\], Cerebroprotective activity \[13\], and Peroxidase activity \[14\], Protective Effect of Red Beetroot against Oxidative Stress \[15\]. The present study was designed to evaluate the anti ulcer effect of ethanolic extract of beet root containing flavonoids\{(+)-Dehydrovomifoliol, 3-Hydroxy-5a,6a-epoxy-b-ionone, Vitexin 7-O-b-D-glucopyranoside, Vitexin 2''-O-b-D-glucopyranoside\} and phenolic compounds on physical and chemical factors induced gastric ulcer models in rats. These flavonoids having anti ulcerogenic property \[16\]. There are no scientific reports available in support of its
traditional claim of anti-ulcer potential of *beta vulgaris* L. Hence, the present study is focused to explore the anti-ulcer properties of *beta vulgaris*.

II. Materials and Methods:

II.1. Plant material:

Beet roots (*Beta vulgaris* L.) were purchased from local markets in Warangal. It was identified and authentificated by Professor Dr. Md. Mustafa, Department of botany, Kakatiya university, Warangal, Telangana.

II.2. Animals:

Healthy wistar albino rats weighing between 200-250g were used for the study. The animals were procured from Sainath agencies, laboratory animals, Hyderabad and the animals were kept in polypropylene cages (6 in each cage) and animals were acclimatized to our lab environment for about a week prior to the study, so that they could adapt to the new environment. Animal house were maintained under standard hygienic conditions, at 25 ± 2°C, humidity (60 ± 10 %) with 12 hrs day and night cycle, with food and water *ad libitum*. The experiments were carried out prior approval from Institutional Animal Ethical Committee (IAEC).

II.3. Extract preparation:

The root pieces were shade-dried and made into a coarse powder which was passed through a 40-mesh sieve to get a uniform particle size and then used for extraction. A weighed quantity (500 g) of the powder was then subjected to continuous hot extraction in Soxhlet apparatus with ethanol and the residual marc was collected. The extract was filtered through a cotton plug, followed by whatman filter paper (no.1). The extract was evaporated under reduced pressure using a rotovac evaporator at a low temperature (40-60°C) until all the solvent had been removed to give an extract sample.

II.4. Acute Toxicity Studies:

The acute toxicity was determined on female albino rats by fixed dose method of OECD Guide line no 420 given by CPCSEA. Groups of 6 rats were administered test drug by oral route at a dose of 2000, 300mg/kg (6 animals in each dose) and mortality was observed after 24 hr. The safe dose was found to be mg/kg body weight. For this study two doses were selected.

II.5. Preliminary phytochemical screening of extracts: Qualitative chemical tests were conducted for ethanolic extracts to identify the various phytoconstituents by employing standard screening tests.
II.6. Models:

a. Alcohol-induced gastric ulcer

The albino rats were randomly divided into four groups of six animals each. Animals were fasted for 24 h before experiment but with free access to water.

**Table no-1. Experimental design of Alcohol-induced gastric ulcer**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>control (1ml of 80% ethanol)</td>
</tr>
<tr>
<td>II</td>
<td>Ranitidine (20mg/kg)</td>
</tr>
<tr>
<td>III</td>
<td><em>Beta vulgaris</em> 250 mg/kg</td>
</tr>
<tr>
<td>IV</td>
<td><em>Beta vulgaris</em> 400mg/kg</td>
</tr>
</tbody>
</table>

**Experimental procedure**

First group treated with 1ml of 80% ethanol orally on the day of experiment at about 10 AM with the help of an oral feeding tube. 2\textsuperscript{nd}, 3\textsuperscript{rd}, 4\textsuperscript{th} groups of animals were treated with ranitidine, low and high doses of beet root extracts respectively one hour before ethanol administration. One hour after drug treatment of 2\textsuperscript{nd}, 3\textsuperscript{rd}, 4\textsuperscript{th} groups of animals were treated with 1 ml of 80% ethanol by p.o, to induce ulcers. The animals were sacrificed after 1hr of ethanol administration. The stomach was opened and calculates the ulcer index and percentage inhibition of ulcer\textsuperscript{[19]}.

b. Aspirin induced ulcer model:

The albino rats were randomly divided into four groups of six animals each. Animals were fasted for 24 h before experiment but with free access to water.

**Table no-2. Experimental design of Aspirin induced ulcer model.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>control (250mg/kg aspirin)</td>
</tr>
<tr>
<td>II</td>
<td>Ranitidine (20mg/kg)</td>
</tr>
<tr>
<td>III</td>
<td><em>Beta vulgaris</em> 250 mg/kg</td>
</tr>
<tr>
<td>IV</td>
<td><em>Beta vulgaris</em> 400mg/kg</td>
</tr>
</tbody>
</table>

**Experimental procedure**

First group treated with Aspirin in a dose of 250 mg/kg was administered orally on the day of experiment at about 10 AM with the help of an oral feeding tube in the form of an aqueous water suspension. 2\textsuperscript{nd}, 3\textsuperscript{rd}, 4\textsuperscript{th} groups of animals were treated with ranitidine, low and high doses of beet root extracts respectively one hour before aspirin administration. One hour after drug treatment of 2\textsuperscript{nd}, 3\textsuperscript{rd}, 4\textsuperscript{th} groups of animals were treated with 250mg/kg aspirin by
p.o, to induce ulcers. The animals were sacrificed after 4hr of aspirin administration. The stomach was opened and calculates the ulcer index and percentage inhibition of ulcer [20].

c. Pylorus- ligation induced gastric ulcer

The albino rats were randomly divided into four groups of six animals each. Animals were fasted for 24 h before experiment but with free access to water.

Table no- 3. Experimental design of Pylorus- ligation induced gastric ulcer

<table>
<thead>
<tr>
<th>Groups</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>control (saline)</td>
</tr>
<tr>
<td>II</td>
<td>Ranitidine (20mg/kg)</td>
</tr>
<tr>
<td>III</td>
<td><em>Beta vulgaris</em> 250 mg/kg</td>
</tr>
<tr>
<td>IV</td>
<td><em>Beta vulgaris</em> 400mg/kg</td>
</tr>
</tbody>
</table>

Experimental procedure

The control group treated with normal saline only. 2nd, 3rd, 4th, groups of animals were treated with ranitidine, low and high doses of beet root extracts respectively one hour before pylorus ligation on the day of experiment at about 10 AM. After 1h of drugs treatment, animals were anaesthetized with the help of anesthetic ether the abdomen was opened by a small midline incision below the xiphoid process. Pyloric portion of the stomach was slightly lifted out and ligated avoiding traction to the pylorus or damage to its blood supply. The stomach was replaced carefully and the abdominal wall was closed by interrupted sutures. Rats were sacrificed by an over dose of anaesthetic ether after four hours of pyloric ligation. The abdomen was opened, cardiac end of the stomach was dissected out and the contents were drained into a glass tube. The volume of the gastric juice was measured and centrifuged at 2000 rpm for 10 min. From the supernatant, aliquots (1 ml of each) were taken for the determination of pH, total and free acidity. Each stomach was examined for lesions in the fore stomach portion and indexed according to severity [21].

II.7. Determination of Ulcer Index (UI)

The ulcerative index was calculated by severity of gastric mucosal lesions and graded as follows; [2]

0=no ulcer, 1=superficial ulcer, 2=deep ulcer, 3=perforation

\[
UI = UN + US + UP \times 10^{-1}
\]

UN=average of number of ulcers per animal

US=average of severity score

UP=Percentage of animals with ulcers
% gastro protection was calculated according to:

\[ \text{% gastro protection} = \frac{(\text{UIC}-\text{UIT})}{\text{UIC}} \times 100 \]

Where, UIC-ulcer index of control. UIT-ulcer index of test

**II.8. Histopathological evaluation**

The gastric tissue was fixed in 10% ethanol buffer formalin and processed through graded ethanol, xylene and impregnated with paraffin wax; sections were made by microtome. After staining with haemotoxylin and eosin stain, the sections were examined under a research microscope by a person who was not aware of experimental protocols. The different histopathological studies screened were: congestion, hemorrhage, edema, necrosis, inflammatory and dysplastic changes, erosions and ulcerations \[22\].

**II.9. Statistical analysis**

The statistical data was expressed as mean ± S. E.M. Statistical analysis was carried out by using one-way analysis of variance (ANOVA) followed by Dunnett’s Multiple Comparison test. Differences between the data were considered significant at \( P<0.05 \) \[2\].

**III. Results**

**III.1. Phytochemical screening:**

The Phytochemical screening of EEBV revealed the presence of Carbohydrates, Glycosides, Proteins & amino acids, Saponins, Tannins & Phenolic compounds and Flavonoids.

**III.2. Acute toxicity studies:**

The ethanolic extract of *beta vulgaris* was subjected for the acute toxicity study to determine the therapeutic dose using albino rats in controlled environment. Acute toxicity study carried out on EEBV up to the dose of 2000 mg/kg demonstrated that the extract did not show any sign of toxicity and mortality. Hence 250 and 400 mg/kg dose of the extract selected for evaluation of anti-ulcer activity.

**III.3. Effect of EEBV on ulcer index and % ulcer protection in ethanol induced gastric ulcer.**

<table>
<thead>
<tr>
<th>Groups (n=5)</th>
<th>Treatment</th>
<th>UI</th>
<th>% ulcer protection</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>11.33±1.732</td>
<td>0.00</td>
</tr>
<tr>
<td>II</td>
<td>Ranitidine 20mg/kg</td>
<td>8.33±0.577**</td>
<td>26.47</td>
</tr>
<tr>
<td>III</td>
<td><em>Beta vulgaris</em> 250 mg/kg</td>
<td>10.76±0.4282ns</td>
<td>5.03</td>
</tr>
<tr>
<td>IV</td>
<td><em>Beta vulgaris</em> 400mg/kg</td>
<td>8.55±0.477**</td>
<td>24.53</td>
</tr>
</tbody>
</table>
Values express as mean ± SEM; n=6 in each group, statistical comparisons as follows: significant at P<0.01 compared to control group, P>0.05 ns-non significant.

Figure no-1. Effect of EEBV on ulcer index in ethanol induced gastric ulcer.

![Graph showing effect of EEBV on ulcer index in ethanol induced gastric ulcer.](image)

Fig no- 2: Macroscopical view of ethanol induced gastric ulcer.

Normal | Toxic control | Standard
---|---|---
![Normal](image) | ![Toxic control](image) | ![Standard](image)

Low dose of beta vulgaris | High dose of beta vulgaris
---|---
![Low dose beta vulgaris](image) | ![High dose beta vulgaris](image)

III.4. Effect of EEBV on ulcer index and % ulcer protection in aspirin induced gastric ulcer.

<table>
<thead>
<tr>
<th>Groups (n=6)</th>
<th>Treatment</th>
<th>Ulcer Index (UI)</th>
<th>% Ulcer Protection</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>11.083±0.4282</td>
<td>0.00</td>
</tr>
<tr>
<td>II</td>
<td>Standard</td>
<td>8.562±0.4216**</td>
<td>22.74</td>
</tr>
<tr>
<td>III</td>
<td>Beta vulgaris 250 mg/kg</td>
<td>10.35±0.5627ns</td>
<td>6.613</td>
</tr>
<tr>
<td>IV</td>
<td>Beta vulgaris 400mg/kg</td>
<td>8.516±0.42816**</td>
<td>23.16</td>
</tr>
</tbody>
</table>
Values express as mean ± SEM; n=6 in each group, statistical comparisons as follows: significant at P<0.01** compared to control group, P>0.05 ns-non significant

**Figure V.3. Effect of EEBV on ulcer index in aspirin induced gastric ulcer.**

![Bar chart showing ulcer index for different groups with control, standard ranitidine, low dose, and high dose.](image)

**Figure V.4. Macroscopical view of aspirin induced gastric ulcer**

![Macroscopic view of aspirin induced gastric ulcer for normal, toxic control, standard, low dose, and high dose groups.](image)

### III.5. Effect of EEBV on ulcer index and % ulcer protection in pylorus ligation induced gastric ulcer.

<table>
<thead>
<tr>
<th>Groups (n=5)</th>
<th>Treatment</th>
<th>UI</th>
<th>% ulcer protection</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Toxic control</td>
<td>11.99±0.6009</td>
<td>0.00</td>
</tr>
<tr>
<td>II</td>
<td>Standard</td>
<td>8.532±0.4944**</td>
<td>28.840</td>
</tr>
<tr>
<td>III</td>
<td>Beta vulgaris 250 mg/kg</td>
<td>10.732±1.138ns</td>
<td>10.49</td>
</tr>
<tr>
<td>IV</td>
<td>Beta vulgaris 400mg/kg</td>
<td>8.66±0.6667**</td>
<td>27.77</td>
</tr>
</tbody>
</table>
Values express as mean ± SEM; n=6 in each group, statistical comparisons as follows: significant at P<0.01** compared to control group, P>0.05 ns-non significant.

**Figure.5. Effect of ethanolic extract of *Beta vulgaris* on ulcer index in pylorus ligation induced gastric ulcer.**

**Figure.6. Macroscopical view of pylorus ligation induced gastric ulcer.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Treat Met</th>
<th>Volume of gastric content</th>
<th>Volume of Gastric juice</th>
<th>PH</th>
<th>Total acidity (mEq/lit)</th>
<th>Free acidity (mEq/lit)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>3.2±0.1291</td>
<td>2.133±0.1022</td>
<td>1.865±0.1018</td>
<td>705±1.478</td>
<td>283±2.171</td>
</tr>
<tr>
<td>II</td>
<td>Standard</td>
<td>1.983±0.113**</td>
<td>0.95±0.08466**</td>
<td>4±0.1238**</td>
<td>44.6±0.2186**</td>
<td>24±0.1880**</td>
</tr>
<tr>
<td>III</td>
<td><strong>EEBV</strong> 250mg/kg</td>
<td>2.433±0.233**</td>
<td>1.4±0.2817*</td>
<td>2.8±0.1932**</td>
<td>162.5±1.315**</td>
<td>66.3±0.4447**</td>
</tr>
<tr>
<td>IV</td>
<td><strong>EEBV</strong> 400mg/kg</td>
<td>2.15±0.1945**</td>
<td>1.183±0.1327**</td>
<td>3.46±0.1936**</td>
<td>52.8±0.2358**</td>
<td>21.76±0.2088**</td>
</tr>
</tbody>
</table>
Above Values express as mean ± SEM; n=6 in each group, statistical comparisons as follows: significant at P<0.01**, p<0.05* compared to control group.

**Figure.7. Histopathological photographs of pylorus ligation induced ulcer in rats**

<table>
<thead>
<tr>
<th>Normal</th>
<th>ulceration</th>
<th>standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low dose</td>
<td>high dose</td>
<td></td>
</tr>
</tbody>
</table>

**Group-I:** (Negative control). Rats were given distilled water orally prior to stomach ulcer induction with pylorus ligation. The histopathological finding revealed numerous erosions, shows deep ulceration of granular epithelium and almost reducing the sub-mucosa and damaged the muscularis propria.

**Group-II:** (Positive control). Animals in this group were given ranitidine (20mg/kg), i.p before ulcer induction. The mucosa was fairly protected but some of areas show slightly 2-3 spot ulcers, more protective effect on sub mucosa and muscularis propria.

**Group-III:** (Low dose of EEBV). The histopathologic effect of ethanolic extract of *beta vulgaris* at 250 mg/kg dose has shown mucosal erosion, the partial healing of ulcer with few inflammatory cells.

**Group-IV:** (High dose of EEBV). Animals in this group were given oral treatment of ethanolic extract of *beta vulgaris* at 400 mg/kg prior to pylorus ligation. The gastric epithelium was fairly protected and shown the healed ulcer. The mucosa shows normal some red coloration appear and no inflammatory cells, more protective effect on sub mucosa and muscularis propria.
IV. Discussion:

The anti ulcer activity of *beta vulgaris* was evaluated by employing aspirin, alcohol, and pylorus ligation induced ulcer models. These models cause the gastric ulcer in humans. Many factors and mechanisms are involved in the ulcerogenesis and gastric mucosal damage. Ethanol induced gastric ulcer was employed to study the cytoprotective effect of the extracts. The ethanol-induced ulcers is predominant in the glandular part of stomach and was reported to stimulate the formation of leukotriene C4 (LTC4), mast cell secretory products and reactive oxygen species resulting in the damage of rat gastric mucosa. Alcohol rapidly penetrates the gastric mucosa causing cell and plasma membrane damage leading to increased intra cellular membrane permeability to sodium and water. The massive intracellular accumulation of calcium represents a major step in the pathogenesis of gastric mucosal injury. This leads to cell death and exfoliation in the surface epithelium [24]. The beet root extracts shows protection in dose dependent manner against characteristic lesions produced by ethanol and reduced values of ulcer index as compared control group suggesting its potent cytoprotective activity.

Aspirin causes mucosal damage by decreasing cytoprotective prostaglandin levels through inhibition of PG synthesis and also results in back diffusion of H+ ions into the gastric mucosa and inhibits the release of mucus [25]. In this model ethanolic extract of *beta vulgaris* was produced its ulcer protective effect by counteracting the inhibition of PG synthesis and enhancing the mucus release. Beet root extract was significantly reducing the ulcer index compare to control group.

Pylorus ligation induced ulcer was used to study the effect of beet root extract on gastric acid secretion and mucus secretion. The ligation of the pyloric end of the stomach causes accumulation of gastric acid in the stomach. This increase in the gastric acid secretion causes ulcers in the stomach. The fasting of rats for 24 h followed by ligation of pyloric end of the stomach, the ulcer index is determined 4 h after pylorus ligation. The lesions produced by this method are located in the lumen region of the stomach. The Ethanolic extract of *beta vulgaris* and ranitidine significantly decreased the total acidity and free acidity; and significantly enhance the PH; this suggests that it having an anti secretory effect. Its antiulcer activity is further supported by histopathological study shows that protection of mucosal layer from ulceration and inflammation. Pylorus ligation induced ulcer control rats shown perforated ulcer, deep ulceration of granular epithelium and almost reducing the sub-mucosa. The Ethanolic extract of *beta vulgaris* at 250 mg/kg dose has shown mucosal erosion, the partial healing of ulcer with few inflammatory cells and the dose 400 mg/kg has shown the healed ulcer, normal mucosa and no inflammatory cells. Beet root extracts have been reported to
possess antioxidant activity [26] and to contain various types of compounds such as flavonoids and polyphenolic compounds, saponins and tannins [27]. The gastroprotective effect exhibited by Ethanolic extract beta vulgaris is speculated to be attributed to its antioxidant property, which in turn could be linked to the presence of flavonoids and polyphenolic compounds, saponins and tannins [28]. These compounds most likely inhibit gastric mucosal injury.

V. Conclusion:

The ethanolic extract of beta vulgaris at the doses of 250mg/kg and 400 mg/kg clearly demonstrated antiulcer property in experimental model of rats.

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