Antiulcer and in vitro antioxidant activities of *Jasminum grandiflorum* L.


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**Abstract**

The study was aimed at evaluating the antiulcer and antioxidant activities of 70% ethanolic extract of leaves of *Jasminum grandiflorum* L. (JGLE). The leaves of *Jasminum grandiflorum* L. (Family: Oleaceae) is used in folk medicine for treating ulcerative stomatitis, skin diseases, ulcers, wounds, corns—a hard or soft hyperkeratosis of the sole of the human foot secondary to friction and pressure (Stedman’s Medical Dictionary, 28th ed. Lippincott Williams & Wilkins, Philadelphia. p. 443), etc., Antiulcerogenic activity of JGLE (100 and 200 mg/kg, b.w., orally) was evaluated employing aspirin + pylorus ligation (APL) and alcohol (AL) induced acute gastric ulcer models and ulcer-healing activity using acetic acid-induced (AC) chronic ulcer model in rats. Both the antisecretory and cytoprotection hypothesis were evaluated. The antioxidant activity of JGLE has been assayed by using in vitro methods like 2,2-diphenyl-1-picrylhydrazylhydrate (DPPH) assay, reductive ability, superoxide anion scavenging activity, nitric oxide scavenging activity and total phenolic content, in order to explain the role of antioxidant principles in the antiulcerogenic activity of the extract. There was a significant (P < 0.01) dose-dependent decrease in the ulcerative lesion index produced by all the three models in rats as compared to the standard drug famotidine (20 mg/kg, b.w. orally). The reduction in gastric fluid volume, total acidity and an increase in the pH of the gastric fluid in APL rats proved the antisecretory activity of JGLE. Additionally, JGLE completely healed the ulcer within 20 days of treatment in AC model as evidenced by histopathological studies. Like antiulcer activity, the free radical scavenging activities of JGLE depends on concentration and increased with increasing amount of the extract. These results suggest that leaves of *Jasminum grandiflorum* possess potential antiulcer activity, which may be attributed to its antioxidant mechanism of action.

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**Keywords**: *Jasminum grandiflorum* L. (Oleaceae); Gastric ulcer; Antiulcer; Antioxidant

1. Introduction

Peptic ulcer is one of the major gastro-intestinal disorders, which occur due to an imbalance between the offensive (gastric acid secretion) and defensive (gastric mucosal integrity) factors (Hoogerwerf and Pasricha, 2006). Consequently, reduction of gastric acid production as well as re-inforcement of gastric mucosal production has been the major approaches for therapy of peptic ulcer disease. As a result, more and more drugs, both herbal and synthetic are coming up offering newer and better options for treatment of peptic ulcer. The type of drugs varies from being proton-pump inhibitor to H₂ antagonist or a cytoprotective agent. At the same time, each of these drugs confers simpler to several side effects like arrhythmias, impotence, gynaecomastia, enterochromaffin-like cell (ECL), hyperplasia and haemopoietic changes (Akthar et al., 1992). There are evidences for the participation of reactive oxygen species in the etiology and pathophysiology of human disease, such as neurodegenerative disorders, inflammation, viral infections, autoimmune gastrointestinal inflammation and gastric ulcer (Repetto and Llesuy, 2002). Drugs with multiple mechanism of protective action, including antioxidant activity, may be highly effective in minimizing tissue injury in human diseases. It has been demonstrated that many drugs and formulations possess potent antioxidant action and are effective in healing experimentally induced gastric ulcers (Dhuley, 1999; George et al., 1999; Goel and Sairam, 2002). *Jasminum grandiflorum* L. (Family: Oleaceae) exhibit a wide ecological range and found extensively all over India. The leaves of *Jasminum grandiflorum* are used in the treatment of odontalgia, fixing loose teeth, ulcerative stomatitis, leprosy, skin diseases, ototolrhoea, otalgia, stangury, dysmenorrhoea, ulcers, wounds and corns (Warrier et
Jasminum grandiflorum leaves were collected from Coimbatore district, Tamil Nadu, India, during the month of June 2005. The plant was identified and authenticated by Dr. G.V.S. Moorthy, Joint Director, Botanical Survey of India, Tamil Nadu Agricultural University, Coimbatore, India, where a voucher specimen (No. BSI/SC/5/23/05-06/Tech-240) of the plant has been kept in the herbarium.

2.2. Preparation of the extract

Fresh leaves were collected, shade-dried and powdered mechanically. About 60 g of the leaf powder were extracted with 600 ml of 70% ethanol by maceration at room temperature for 4 h using a mechanical shaker. The extract was dried at 40 °C under vacuum and the yield of the extract was 24%.

2.3. Phytochemical screening

Preliminary phytochemical screening of the powdered leaves was performed for the presence of alkaloids, phenolics, flavonoids, saponins, carotenoids, carbohydrates and glycosides (Khandelwal, 2004).

2.4. Animals

Albino rats of Wistar strain of either sex weighing between 150 and 200 g were used. They were housed in standard cages at room temperature (25 ± 2 °C) and provided with food and water ad libitum. The animals were deprived of food for 24 h before experimentation, but had free access to drinking water. The study was conducted after obtaining institutional ethical committee clearance bearing the number 817/04/ac/CPCSEA.

2.5. Drugs and chemicals

Aspirin was obtained from German Remedies Ltd., Mumbai, India and famotidine from Glenmark Pharmaceuticals Ltd., Mumbai. 2,2-Diphenyl-1-picrylhydrazyl hydrate and nitro blue tetrazolium were obtained from HiMedia Laboratories Pvt. Ltd., Mumbai, India. All other chemicals used in this study were obtained commercially and were of analytical grade.

2.6. Acute toxicity studies

Rats were kept overnight fasting prior to drug administration. A total of five animals were used which received a single oral dose (2000 mg/kg, b.w.) of Jasminum grandiflorum leaf extract (JGLE). After the administration of JGLE, food was withheld for further 3–4 h. Animals were observed individually at least once during the first 30 min after dosing, periodically during the first 24 h (with special attention during the first 4 h) and daily thereafter for a period of 14 days. Once daily cage side observations included changes in skin and fur, eyes and mucous membrane (nasal) and also respiratory rate, circulatory (heart rate and blood pressure), autonomic (salivation, lacrimation, perspiration, piloerection, urinary incontinence, and defecation) and central nervous system (ptosis, drowsiness, gait, tremors and convulsion) changes. Mortality, if any, was determined over a period of 2 weeks (OECD, 2002).

2.7. Selection of dose of the extract

LD50 was done as per OECD guidelines for fixing the dose for biological evaluation. The LD50 of the extract as per OECD guidelines falls under class four values with no signs of acute toxicity at 2000 mg/kg. The biological evaluation was carried out at doses of 100 and 200 mg/kg body weight.

2.8. Antiulcer activity

2.8.1. Aspirin + pylorus ligation-induced ulcer model

JGLE, aspirin and standard antiulcer drug, famotidine (Dharmani et al., 2005; Gupta et al., 2005) were prepared in 0.5% sodium carboxy methyl cellulose (CMC) suspension as vehicle and administered orally once daily at a volume of 10 ml/kg body weight. The animals were divided into four groups, consisting of six each. Group I received aspirin alone (200 mg/kg, p.o.). Groups II and III received JGLE orally at the doses of 100 and 200 mg/kg body weight respectively for 7 days. Group IV received famotidine orally at the dose of 20 mg/kg body weight for 7 days (Yesilada et al., 1997). From days 5 to 7, animals of all the groups received aspirin orally as an aqueous suspension at a dose of 200 mg/kg, 2 h after the administration of respective drug treatment (Goel et al., 1986; Venkataramanappa et al., 1998). Animals in all the groups were fasted for 18 h after the respective assigned treatment and were anaesthetised with anaesthetic ether. The abdomen was opened by a small midline incision below the xiphoid process and pylorus portion of stomach was lifted out and ligated (Shay et al., 1945). Precaution was taken to avoid traction to the blood supply. The stomach was sutured with interrupted sutures. Four hours after pylorus ligation the rats were sacrificed and the stomach was removed. The gastric contents were collected, centrifuged and the volume of the supernatant was expressed as ml/100 g body weight. Free and total acidity were determined by titrating with 0.01N NaOH using Topfer’s reagent and phenolphthalein as indicator (Parmar et al., 1984). The free and total acidity were expressed as μequiv./100 g/h. The stomach was then incised along the greater curvature and observed for ulcers. The number of ulcers was counted using a magnifying glass and the diameter of the ulcers were measured using vernier calipers. The following arbitrary scoring system (Dharmani et al., 2005) was used to grade the incidence and severity of lesions: (i) score 10=denuded

al., 1995). The leaves of this species have a distinction of being used in Indian folk medicine for treating ulcers. Literature suggests the use of this plant as a diuretic and spasmodic agent, which is given during childbirth (Somanadhan et al., 1998; Lis-Balchin et al., 2002).

The objective of the present study was to investigate the antiulcer and antioxidant activities of the ethanolic extract of the leaves of Jasminum grandiflorum using various models.
epithelium; (ii) score 20 = petechial and flank haemorrhages; (iii) score 30 = one or two ulcers; (iv) score 40 = multiple ulcers; (v) score 50 = perforated ulcer.

Ulcer index (UI) was then calculated from the above scorings as follows:

\[ \text{UI} = \frac{U_N + U_s + U_p}{10} \times 10^{-1} \]

where \( U_N \) is the average of number of ulcers per animal, \( U_s \) is the mean severity of ulcer score and \( U_p \) is the percentage of animals with ulcer incidence.

2.8.2. Absolute alcohol-induced ulcer

The animals were divided into five groups, consisting of six each. Group I received alcohol alone (1 ml of absolute alcohol, p.o.). Groups II and III received JGLE orally at the doses of 100 and 200 mg/kg body weight, respectively. Group IV received famotidine 20 mg/kg body weight orally (Yesilada et al., 1997; Gupta et al., 2005). JGLE and famotidine were administered orally 30 min before the oral administration of 1 ml of absolute alcohol (Dharmani et al., 2004). Sixty minutes later, the animals were sacrificed and their stomachs excised and gastric contents were aspirated. Stomachs were removed and kept immersed in 10% formalin for 5 min. Each stomach was incised along the greater curvature and examined for linear haemorrhagic lesions in the glandular region. The length (mm) of each lesion was determined at 10× magnification with pair of dividers and each length was summed per stomach. The sum of length (mm) of all lesions for each stomach was used as the ulcer index (UI). Stomachs were again immersed in 10% formalin for 24 h and histopathological examinations were carried out and later the slides were photographed. The percentage inhibition was calculated by the following formula:

\[ \% \text{ inhibition} = \left( \frac{\text{UI treated} - \text{UI control}}{\text{UI control}} \right) \times 100 \]

2.8.3. Acetic acid-induced gastric ulcers

Rats were starved for 24 h prior to the experiment and were divided into four groups, one for control and the other three for drug treatment. Under light ether anaesthesia, laparotomy was performed through a midline gastric incision. After exposing the stomach, 0.05 ml of 10% acetic acid was injected into the subserosal layer in the glandular part of the anterior wall (Okabe and Pfeiffer, 1972). The stomach was bathed with saline to prevent adhesion to the external surface of the ulcerated region. The vehicle, extract and standard drug was administered orally for 20 days beginning one day after surgery. On the 21st day the animals were sacrificed at proper intervals to assess the healing processes of the ulcer. The stomach was removed and the gastric lesions were evaluated by examining the inner gastric surface with a dissecting binocular microscope. Subsequently the ulcer area (mm²) and curative rate % were determined.

2.8.4. Histopathological evaluation of acetic acid-induced ulcers

Stomachs were immersed in a 10% formalin solution for histopathological examination following the assessment of ulcer score. The central part of the damaged (or) ulcerated tissue (if present) was cut in half along the long diameter. If the stomach was protected from the damage then the section was taken from the basal part. After the standard processing, the wet tissue was embedded in paraffin and cut into 5 µm thick section in a rotary microtome. The sections were stained with haematoxylin-eosin and mounted with Canada balsam. These were examined under the microscope for histopathological changes such as oedema, inflammation, infiltration and erosion and photographs were taken.

2.9. In vitro antioxidant activity

2.9.1. DPPH assay

The free radical scavenging activity of JGLE was measured in vitro by 1,1-diphenyl-2-picryl-hydrazyl (DPPH) assay using the method of Blois (1958). About 0.3 mM solution of DPPH in 100% ethanol was prepared and 1 ml of this solution was added to 3 ml of the extract dissolved in ethanol at different concentrations (10–50 µg/ml). The mixture was shaken and allowed to stand at room temperature for 30 min and the absorbance was measured at 517 nm using a spectrophotometer. The IC50 value of the crude extract was compared with that of ascorbic acid, which was used as the standard.

2.9.2. Reductive ability

The reducing power of JGLE was investigated by the Fe³⁺–Fe²⁺ transformation in the presence of the extract using the Oyaizu (1986) method. One millilitre of the plant extract (10–100 µg/ml), 2.5 ml of phosphate buffer (pH 6.6) and 2.5 ml of 1% potassium ferricyanide were incubated at 50 °C for 30 min and 2.5 ml of 10% trichloroacetic acid were added to the mixture and centrifuged for 10 min at 3000 × g. About 2.5 ml of the supernatant was diluted with 2.5 ml water and is shaken with 0.5 ml of freshly prepared 0.1% ferric chloride. The absorbance was measured at 700 nm. Butylated hydroxy toluene (BHT) was used as the standard. All tests were performed in triplicate and the graph was plotted with the average of the three determinations.

2.9.3. Scavenging of superoxide anion radical

The superoxide radical generated by hypoxanthine and xanthine oxidase system was determined by the method described by Guzman et al. (2001). A reaction mixture with a final volume of 1 ml per tube was prepared with 50 mM KH₂PO₄–KOH pH 7.4 containing 1 mM EDTA, 100 µl hypoxanthine, 100 µM nitro blue tetrazolium (NBT), 0.666 U per tube of xanthine oxidase and 10 µl of saline. Xanthine oxidase was added at last. The reaction mixtures were incubated at 25 °C for 5 min, and the absorbance was measured at 560 nm. Ascorbic acid was used as the standard. The results are expressed as the percentage inhibition of NBT reduction rate with respect to the reaction mixture without test compound. All tests were performed in triplicate.

Percentage inhibition was calculated using the formula:

\[ I(\%) = \frac{A_0 - A_I}{A_0} \times 100 \]
where $A_0$ is the absorbance of the control and $A_1$ is the absorbance of the test compound.

### 2.9.4. Determination of total phenolic content

Total soluble phenolic compounds of the JGLE were determined with Folin Ciocalteu reagent (Gulcin et al., 2002). A 0.1 ml aliquot of a suspension of 1 mg of extract in water was totally transferred to a 100 ml Erlenmeyer flask and the final volume was adjusted to 46 ml by the addition of distilled water. Folin Ciocalteu reagent (1 ml) was added to this mixture, followed by 3 ml 2% sodium carbonate 3 min later. Subsequently, the mixture was shaken for 2 h at room temperature and absorbance measured at 760 nm. All tests were performed in triplicate. The concentration of total phenolic compounds in JGLE was determined as $\mu$g pyrocatechol equivalents by using an equation obtained from standard pyrocatechol graph:

$$\text{absorbance} = 0.001 \times \text{pyrocatechol (}\mu\text{g}) + 0.0033.$$

### 2.9.5. Assay of nitric oxide scavenging activity

This experiment was performed according to the method with some modification as described by Sreejayan and Rao (1997). Nitric oxide generated from sodium nitroprusside in aqueous solution at physiological pH interacts with oxygen to produce nitric ions, which were measured by Griess reagent. The reaction mixture (3 ml) containing 10 mM sodium nitroprusside in phosphate buffered saline, and the extract and the reference compound (curcumin) at different concentrations (10, 25, 50, 75, 100 $\mu$g/ml) were incubated at 25 $^\circ$C for 150 min. 0.5 ml aliquot of the incubated sample was removed at 30 min intervals and 0.5 ml Griess reagent was added. The absorbance of the chromophore formed was measured at 546 nm. Inhibition of the nitric oxide generated was measured by comparing the absorbance values of control, extract and curcumin.

### 2.10. Statistical analysis

All the in vivo experimental results were expressed as mean ± S.E.M. Data were analysed by analysis of variance (ANOVA) followed by Dunnett’s test, with the level of significance set at $P<0.01$. All the in vitro experimental results were expressed as mean ± S.E.M. of three parallel measurements. $P$ values < 0.01 were considered significant.

### 3. Results

#### 3.1. Phytochemical screening

Phytochemical screening of the powdered leaves showed the presence of phenolics, flavanoids and carotenoids.

#### 3.2. Acute toxicity studies

In LD$_{50}$ studies, it was found that the animals were safe up to a maximum dose of 2000 mg/kg body weight. There were no changes in normal behaviour pattern and no signs and symptoms of toxicity and mortality were observed. The biological evaluation was carried out at doses of 100 and 200 mg/kg body weight.

#### 3.3. Aspirin + pylorus ligation-induced ulcer model

Animals in the APL group showed a significant ($P<0.01$) increase in the ulcer index and acid secretory parameters like gastric volume, pH, free and total acidity when compared with those of vehicle treated group. In the rats of this group, a number of perforated ulcers (score 50) were also observed. Administration of JGLE produced significant ($P<0.01$) decrease in ulcer index in a dose dependent manner. All the ulcers were of scores 10 and 20 and no perforated ulcers were observed. The extract also significantly reduced the gastric volume, total and free acidity, and increased the pH of the gastric fluid, proving its antisecretory activity. JGLE at a dose of 100 and 200 mg/kg body weight showed protection index of 53% and 64%, respectively, where as famotidine showed protection index of 69% at a dose of 20 mg/kg body weight (Table 1).

#### 3.4. Absolute alcohol-induced ulcer model

Administration of ethanol produced haemorrhagic gastric lesions in the gastric mucosa of the control group. JGLE reduced these lesions as evidenced by a significant ($P<0.01$) reduction in the ulcer index when compared with the control group. JGLE at a dose of 100 and 200 mg/kg body weight showed protection

### Table 1

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg)</th>
<th>Gastric volume (ml/100 g)</th>
<th>pH</th>
<th>Free acidity (µequiv./100 g/4 h)</th>
<th>Total acidity (µequiv./100 g/4 h)</th>
<th>Ulcer index</th>
<th>% inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>200 (p.o.)</td>
<td>2.32 ± 0.05</td>
<td>1.55 ± 0.013</td>
<td>77.33 ± 0.52</td>
<td>95.03 ± 1.12</td>
<td>32.83 ± 0.85</td>
<td>–</td>
</tr>
<tr>
<td>JGLE</td>
<td>100 (p.o.)</td>
<td>0.77 ± 0.01*</td>
<td>3.55 ± 0.01*</td>
<td>36.55 ± 0.05*</td>
<td>42.76 ± 0.71*</td>
<td>15.51 ± 0.76*</td>
<td>53</td>
</tr>
<tr>
<td>JGLE</td>
<td>200 (p.o.)</td>
<td>0.54 ± 0.01**</td>
<td>4.41 ± 0.13**</td>
<td>17.55 ± 0.41**</td>
<td>31.12 ± 0.42**</td>
<td>10.95 ± 0.44**</td>
<td>64</td>
</tr>
<tr>
<td>Famotidine</td>
<td>20 (p.o.)</td>
<td>0.44 ± 0.03**</td>
<td>5.06 ± 0.08**</td>
<td>15.40 ± 0.38**</td>
<td>25.71 ± 1.0**</td>
<td>10.21 ± 0.26**</td>
<td>69</td>
</tr>
<tr>
<td>$F$</td>
<td>722</td>
<td>165.86</td>
<td>385.3</td>
<td>284.23</td>
<td>121.71</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$P$</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as the mean ± S.E.M.; $n = 6$ in each group. *$P<0.05$, **$P<0.01$ when compared to aspirin treated group (one-way ANOVA followed by Dunnett’s test).
Table 2
Effect of *Jasminum grandiflorum* leaf extract (JGLE) on absolute alcohol-induced gastric ulcer in rats

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg)</th>
<th>Ulcer index</th>
<th>% ulcer inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>–</td>
<td>29.86 ± 0.787</td>
<td>–</td>
</tr>
<tr>
<td>JGLE</td>
<td>100 (p.o.)</td>
<td>9.93 ± 0.330</td>
<td>66</td>
</tr>
<tr>
<td>JGLE</td>
<td>200 (p.o.)</td>
<td>8.59 ± 0.199**</td>
<td>71</td>
</tr>
<tr>
<td>Famotidine</td>
<td>20 (p.o.)</td>
<td>8.25 ± 0.316**</td>
<td>73</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± S.E.M.; *n* = 6 in each group. *F* values: *F*(3,20) = 54.2 (*P* < 0.01) for absolute alcohol. *P* < 0.05, **P** < 0.01 (one-way ANOVA followed by Dunnett’s test).

Table 3
Effect of *Jasminum grandiflorum* leaf extract (JGLE) on acetic acid-induced gastric ulcer in rats

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose (mg/kg)</th>
<th>Ulcer index</th>
<th>% ulcer inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>–</td>
<td>29.32 ± 0.597</td>
<td>–</td>
</tr>
<tr>
<td>JGLE</td>
<td>100 (p.o.)</td>
<td>13.83 ± 0.492*</td>
<td>56</td>
</tr>
<tr>
<td>JGLE</td>
<td>200 (p.o.)</td>
<td>10.31 ± 0.42**</td>
<td>66</td>
</tr>
<tr>
<td>Famotidine</td>
<td>20 (p.o.)</td>
<td>8.88 ± 0.24**</td>
<td>72</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± S.E.M.; *n* = 6 in each group. *F* values: *F*(3,20) = 50.7 (*P* < 0.01) for acetic acid model. *P* < 0.05, **P** < 0.01 (one-way ANOVA followed by Dunnett’s test).

The index of 66% and 71%, respectively. Famotidine also significantly (*P* < 0.01) reduced ulcer index of ethanol-induced gastric ulcers (Table 2).

3.5. Acetic acid-induced gastric ulcer model

Treatment with JGLE at a dose of 100 and 200 mg/kg body weight significantly (*P* < 0.01) healed ulcers after 20 days of treatment showing protection index of 56% and 66%, respectively, where as famotidine showed protection index of 72% (Table 3). Complete healing of ulcers was observed after 20 days of treatment.

3.6. Histopathology of acetic acid-induced ulcers

After 20 days of treatment, the rats treated with acetic acid showed loss of gland architecture with erosion of the epithelial layer and evident oedema and infiltration by inflammatory cell (Fig. 1). JGLE (100 mg/kg) treated rats showed no ulceration but intactness of gastric epithelium was not completely restored. Minimal oedema and infiltration was seen in the lower half of the mucosa (Fig. 2). JGLE (200 mg/kg) treated rats showed no ulceration in the mucosa. Glands are regular with complete restoration of gastric epithelium. Minimal oedema and infiltration were seen in one area (Fig. 3). Famotidine treated groups showed no ulceration in gastric mucosa, glands were regular and no inflammation was observed (Fig. 4).

3.7. In vitro antioxidant activity

JGLE reduced DPPH to a yellow coloured product in a concentration dependent manner. The IC$_{50}$ value of the crude extract was compared with that of ascorbic acid, which was used as standard. It is found that 15 μg/ml of the extract was as potent as 12 μg/ml of ascorbic acid. This shows a good free radical scavenging activity of JGLE. The reducing power of the extract was compared with that of standard, butylated hydroxy toluene. The increase in absorbance of the reaction mixture containing the extract showed increased reducing power with increase in concentration. The IC$_{50}$ value for the extract was found to be 19.5 μg/ml where as IC$_{50}$ value for quercetin is 15.5 μg/ml. The reduction of NBT was found to increase in a dose dependent manner. The scavenging ability of phenol is due to their hydroxyl group, which contribute directly to its antioxidant activity. In 4 μg/ml of JGLE, 3.61 μg pyrocatechol equivalent was detected. The nitric oxide radical produced from sodium nitroprusside at physiological pH were inhibited by JGLE and was found to be 98.0 μg/ml. The IC$_{50}$ value for curcumin is 92 μg/ml.
4. Discussion

Peptic ulcer results due to overproduction of gastric acid (or) decrease in gastric mucosal production. Aspirin + pylorus ligation (APL)-induced ulcers occur because of an increase in acid-pepsin accumulation due to pylorus obstruction and subsequent mucosal digestion (Goel and Bhattacharya, 1991). Further, the role of free radicals is also reported in the induction of ulcers. Aspirin-induced ulcer is mediated through tissue damaging free radicals (Scheiman, 1996), which are produced from the conversion of hydroperoxyl to hydroxy fatty acids, which leads to cell destruction. The hydroperoxyl fatty acids are generated from the degeneration of mast cells and generalized lipid peroxidation accompanying cell damage (Van Kolschoten et al., 1983). In folk medicine, *Jasminium grandiflorum* leaves have been used for the treatment of ulcerative stomatitis, ulcers, wounds, corns, etc.

The present study reveals that JGLE treated groups showed a significant ($P < 0.01$) increase in gastric juice pH, reduces the gastric volume, free acidity and total acidity when compared to control. This effect was similar to famotidine treated group. JGLE decreased the ulcer index more effectively in a dose-dependent manner. These results show that the antiulcer activity of JGLE might be due to its antisecretory activity.

Ethanol-induced gastric ulcers have been widely used for the evaluation of gastroprotective activity. Ethanol is metabolised in the body and releases superoxide anion and hydroperoxy free radicals. It has been found that oxygen-derived free radicals are implicated in the mechanism of acute and chronic ulceration in the gastric mucosa (Pihan et al., 1987) and scavenging these free radicals can play an appreciable role in healing these ulcers (Halliwell and Gutteridge, 2001). JGLE significantly ($P < 0.01$) reduced the ulcer index and afforded significant protection against ethanol-induced ulcer. The antioxidant properties of JGLE may have scavenged the free radicals produced by the metabolism of ethanol and thereby heal the ulcers.

Acetic acid-induced chronic ulcer model was chosen because it produces gastric lesion, which is similar to human chronic ulcers. In this model, acetic acid produced mucosal injury, which was confined to the glandular stomach (Dharmani et al., 2004). The ulcer produced by acetic acid is due to the release of histamine, which increases the capillary permeability and back diffusion of HCl (Takagi et al., 1969). Treatment with JGLE for 20 days afforded complete regeneration of mucosal glandular structure, which was evidenced through histopathological studies of the stomach.

Reactive nitrogen species (RNS) has been suggested to be involved in gastric mucosal damage (Halliwell and Gutteridge, 2001). The extract showed a dose-dependent inhibition of nitric oxide free radicals. The RNS scavenging activity of the extract may be contributed to the presence of phenolic compounds, which is also proved by Folin–Ciocalteu method.

The results of our study prove that the crude extract of *Jasminium gradiflorum* possess antiulcer activity against experimentally induced acute and chronic gastric ulcer models. Hence, it can be suggested that the antiulcer activity of the extract may be attributed to its antisecretory and antioxidant activities.

References


