Evaluation of Anti-Ulcer Activity of Arisaema Leschenaultii

Vlnay Jain¹ and M.K. Panigrahi²

Abstract
Ethanolic and Aqueous extract of Arisaema Leschenaultii was evaluated for its anti-ulcer activity against naproxen induced ulcer. Different extracts of blume of A. leschenaultii gave dose dependent increase in anti-ulcer activity against naproxen induced ulcer. Ethanolic extract of blume of A. leschenaultii showed better ulcer inhibition (55.8%) as compared to AEAL (36.5%). The present investigation revealed that Arisaema Leschenaultii exhibited significant antiulcer activity by enhancing antioxidant potential of gastric mucosa thereby reducing mucosal damage.

Keywords: Anti-ulcer activity, Herbal drug, Antioxidant,

Introduction
Arisaema leschenaultii (B.) AL. (Family Araceae) is commonly known as Dhei or Cobra Lilly. It is widely distributed over the greater part of India on the hills of Assam, Karnataka, Kerala and Tamilnadu. Different parts of plant are traditionally used in Ayurveda for the treatment of urinary diseases, colitis, eczema, purging, gonorrhea, piles, haemorrhoids, syphills, roundworm, fistula and sinus [1]. The whole plant of this species has been reported to show antiseptic property in buffaloes. Kumari et al., studied that AL is used as abortifacient and contraceptives for pig and cattle and also reported the method of preparing contraceptives from this plant[2]. Satyanarayanan reported the fructosans is present in AL [3].

Material and methods
Extraction Of Drug Material
Plant material of A. leschenaultii was collected from different regions, thoroughly washed, and dried at 55°C in an air dryer for 48 h. Dried plant parts were powdered with Wiley Mill (Model 4276-M, Thomas Scientific, USA) to pass 20 mesh sieve and stored in sealed plastic bags. About 200 g of powdered material was taken and extracted with different techniques of extraction (soxhlet extraction, percolation, maceration, sonication, homogenization and microwave extraction) using different solvents (petroleum ether, benzene, chloroform, acetone, ethanol, methanol and water). Each process was repeated thrice for complete extraction. After extraction, extracts were combined and evaporated to dryness in vacuo.

Anti-ulcer

The present study deals with anti-ulcer activity of A. leschenaultii extracts in naproxen induced ulcer. The experimental setup of the study was given below.

Experimental setup
Group 1: Control group (10 mL/kg/day of saline)
Group 2: Omeprazole (30 mg/kg p.o.)
Group 3: EEAL (100 mg/kg/day in 1% CMC, p.o.)
Group 4: EEAL (200 mg/kg/day in 1% CMC, p.o.)
Group 5: AEAL (100 mg/kg/day in 1% CMC, p.o.)
Group 6: AEAL (200 mg/kg/day in 1% CMC, p.o.)

Naproxen induced ulcer
One hour after drug treatment gastric ulcer was induced by the modified procedure [4]. Control was administered with 10 mL/kg of normal saline. Ethanolic and aqueous extracts (100 and 200 mg/kg) were given to test groups. Omeprazole (30 mg/kg p.o.) was used as standard. One hour later naproxen (30 mg/kg p.o.) was administered. Animals were sacrificed after six hours of naproxen administration, stomach was isolated and opened along greater curvature to expose inner surface. Inner surface was washed thoroughly with normal saline. The ulcer area, ulcer index and percentage inhibition were calculated by using image analysis software [5].

Result and discussion

Anti-ulcer activity
The present study deals with anti-ulcer activity of A. leschenaultii extracts in naproxen induced ulcer. Naproxen is a non-steroidal anti-inflammatory drug (NSAID), which can directly damage the gastric epithelium by intracellular accumulation of drug in an ionised state and reduce the hydrophobicity of the mucus gel layer by changing the action of surface active phospholipids [6]. The
enzymes such as catalase and glutathione peroxidase provide defence against damage of gastric mucosa after administration of NSAIDs and also decrease lipid peroxide level in rats [7 & 5]. The ethanolic extracts of A. leschenaultii reduced lipid peroxide level by scavenging free radical and might increase the activity of anti-oxidant enzymes (catalase and glutathione peroxidase).

Neutrophil adherence to the endothelium of gastric microcirculation is critical in NSAID injury [8]. Neutrophil adherence damages the mucosa by liberating oxygen free radicals, releasing proteases and obstructing capillary blood flow. NSAIDs might induce the synthesis of tumour necrosis factor (TNFα) and leukotrienes and these inflammatory mediators stimulate neutrophil adherence by up-regulation of adhesion molecules [9, 10 & 11]. The free radical scavenging effect, anti-TNFα activity, prostaglandins like protective action and leukotrienes inhibition by A. leschenaultii extracts might reverse the effect of neutrophil adherence.

Gastric acid probably exacerbates NSAID injury by disrupting the basement membrane to produce deep injury, affecting platelet aggregation and impairing ulcer healing [12, 13 & 14]. Ethanolic extracts of A. leschenaultii is effective against first phase of inflammation, which occurs due to the release of histamine, serotonin and kinins. Thus, A. leschenaultii might reduce the secretion of gastric acid by blocking histamine receptor.

Ulcer healing is a complex process that involves combination of wound retraction and re-epithelialization. It also involves growth factors and angiogenesis [15]. A. leschenaultii significantly reduced the size of ulcer.

Table 1 shows the effect of A. leschenaultii extracts on naproxen induced ulcer. Our result showed that different extract of blume of A. leschenaultii gave dose dependent increase in anti-ulcer activity against naproxen induced ulcer. Ethanolic extract of blume of A. leschenaultii showed better ulcer inhibition (55.8%) as compared to AEAL (36.5%). The results were significant as compared to control (P<0.05).

The anti-ulcer activity of A. leschenaultii extracts might be due to antioxidant, anti-secretory, protective action and leukotrienes inhibition. The morphological representation of anti-ulcer activity of different extracts of A. leschenaultii is presented in Figure 1.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Dose (mg/kg)</th>
<th>US (mm²)</th>
<th>UI</th>
<th>% I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>-</td>
<td>7.34±0.22</td>
<td>1.23±0.04</td>
<td>-</td>
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<tr>
<td>Omeprazole</td>
<td>30</td>
<td>2.54±0.09**</td>
<td>0.37±0.01**</td>
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<tr>
<td>EEAL</td>
<td>100</td>
<td>4.72±0.01**</td>
<td>0.73±0.01**</td>
<td>44.20</td>
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<tr>
<td></td>
<td>200</td>
<td>4.05±0.52**</td>
<td>0.66±0.08*</td>
<td>55.80</td>
</tr>
<tr>
<td>AEAL</td>
<td>100</td>
<td>5.87±0.12</td>
<td>0.95±0.02*</td>
<td>31.10</td>
</tr>
<tr>
<td></td>
<td>200</td>
<td>5.36±0.13**</td>
<td>0.88±0.06*</td>
<td>36.50</td>
</tr>
</tbody>
</table>

US: Ulcer surface; UI: Ulcer index; % I: Percent inhibition.

All values are expressed as mean ± SEM (n=6); One-way ANOVA followed by Dunnett's test; *P<0.05 and **P<0.01 considered significant as compared to control.
References


