TO STUDY ANALGESIC AND ANTI-INFLAMMATORY EFFECT OF KARANJA (PONGAMIA PINNATA PIERRE) SEED CHURANA IN SWISS ALBINO MICE

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ABSTRACT

Objectives: The present study was carried out to evaluate 1) anti-inflammatory activity 2) analgesic activity, of the Karanja (Pongamia pinnata Pierre) Seed Churana Methods: To study anti-inflammatory activity carrageenin was used. The study was conducted in control group and test drug group at the dose 800mg and 1600mg/kg body weight. To study analgesic activity acetic acid induced writhing test was performed and values were observed in control and test drug group at different doses. Results: An apparent suppression of oedema formation was observed at both the dose levels. At 800 mg/kg dose level, 11.81% suppression of oedema was observed and it was statistically non-significant. At higher dose level (1600 mg/kg), the suppression was 40.39% and it was statistically significant (p < 0.05). In lower test drug dose group (2.5 g/kg), 34.06% decrease in number of acetic acid induced writhing was observed, while in higher test drug dose group (5.0 g/kg) 30.31% decrease was observed. However, both changes were found to be statistically non-significant. Conclusion: Karanja seed possess significant anti-inflammatory and weak peripheral analgesic activity.

KEYWORDS: Karanja, anti-inflammatory, analgesic, carrageenin, writhing.

INTRODUCTION

The international association of study of pain definition states “pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.”[1] Pain is a major symptom in many medical conditions and significantly interferes with a person’s quality of life and general functioning. Analgesics are drugs that selectively relieve pain by acting on the central nervous system or peripheral pain mechanisms without altering the consciousness.

Inflammation is the complex biological response of vascular tissues to harmful stimuli, such as pathogens, damaged cells, or irritants. It is a complex reaction in tissues that consists mainly of response of blood vessels and leukocytes. It is a series of host responses directed as a protective attempt by the organism to remove the injurious stimuli as well as initiate the healing process for the tissue.[2] Most of the drugs used at present for analgesic effect are synthetic in nature and prolonged use of these drugs causes severe side-effects.[3] In this context, there arises new scope for evaluation of herbs for treatment of pain. Plants still remain a large untapped source of structurally novel compounds that might serve as a lead for the development of novel drugs.

Pongamia pinnata Pierre known as Karanja is one of the herbs mentioned in all ancient scriptures of Ayurveda. The plant found throughout India upto 1300 metres altitude, along the streams and river, mainly in Andhra Pradesh, Kerala, Maharashtra and Tamil Nadu. It is an evergreen, medium sized glabrous tree. It is one of the best herbs in skin diseases. Also used in whooping cough, hepato splenomegaly, piles, worm infestation, piles, anorexia and liver diseases.[4]

AIMS AND OBJECTIVES

1. To evaluate the test drug for anti-inflammatory activity,
2. To evaluate the test drug for analgesic activity.

MATERIALS AND METHODS

Drug material
The drug Pongamia pinnata Pierre, seed powder (churana) sample was supplied by Pharmacy, Gujarat Ayurveda University, Jamnagar and identified and authenticated in Pharmacognosy laboratory. For administering to the experimental animals, a drug suspension was made with requisite quantity of distilled water according to the dose required.
Animals
Swiss albino mice and Charles Foster strain albino rats were obtained from the animal house attached to the I.P.G.T.R.A, Gujarat Ayurveda University, Jamnagar. The animals were maintained on Navchakan Oil Mills, "Amrut" Brand rat pellets and tap water given ad libitum. The animals were maintained under normal ambient conditions. Each experimental group consisted 6 rats of either sex. Control group received equal quantity of the Vehicle (distilled water) used for the preparation of the test drug suspension.

Chemicals
Carrageenin and acetic acid 3% v/v were used for anti-inflammatory and analgesic activity respectively.

Instruments used
Plethysmograph, weighing scale, monopane balance, incubator, sterilizer, surgical instruments, cotton, syringes, needles, centrifuge, refrigerator, feeding syringes and tubes, serological water bath.

Route of administration
The drug was administered by oral route with the help of a gastric catheter sleeved on to a syringe. The dose of the drug was calculated by extrapolating the human dose to animals based on the body surface area ratio by referring to the standard table of Paget and Barnes (1994). [4]

Statistical analysis
Student’s t-test for unpaired data has been used for analyzing the data generated during the study. [5]

ANTI INFLAMMATORY ACTIVITY
Carrageenin induced paw oedema in rats [6]
Aqueous solution of Pongamia pinnata seeds was screened for anti-inflammatory activity against carrageenin induced hind paw oedema in rats. The method described by Winter et al was followed. It is the basic test for screening anti-inflammatory effect. In this method paw volume is measured plethysmographically before and three hours after carrageenin injection. It produces marked swelling of the paw and anti-inflammatory drugs are supposed to suppress this swelling.

Procedure
Rats of either sex weighing between 180-300 g were used. The test drug was administered at a dose of 800 mg and 1600 mg per Kg body weight in two groups for seven days, while in control group equal volume of tap water was administered. On eighth day, initially left hind paw volumes upto the tibio-tarsal articulation were recorded by using plethysmograph. The test drug was administered in accordance with body weight of rats. The rats were administered 2 ml per 00 g body weight of tap water to ensure uniform hydration and hence to minimize variations in oedema formation. 60 minutes after the administration of the drug, paw oedema was induced by injecting 0.1 ml of 1% carrageenan aqueous suspension in normal saline into the plantar aponeurosis of left hind limb. Following Bhatt et al (1977) procedure the left hind paw volume was recorded three hours after carrageenin injection by using plethysmograph. Results were expressed as an increase in paw volume in comparison to the initial volumes and also in comparison with control group. If the percentage in paw volume is significantly less in test drug administered group in comparison to control group, then the drug was considered to possess anti-inflammatory activity.

RESULTS AND OBSERVATIONS
Data on the effect of test drug on 1% carrageenin induced hind paw oedema in rats have been summarized in table 1.

Table 1: Effect of Karanja Seed Churana on on Carrageenin induced hind paw oedema in albino rats.

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose (mg/kg)</th>
<th>Percentage increase in paw volume</th>
<th>Percentage decrease in paw volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>-</td>
<td>85.66±10.53</td>
<td>-</td>
</tr>
<tr>
<td>Karanja seed churana</td>
<td>800</td>
<td>75.55±8.05</td>
<td>11.81</td>
</tr>
<tr>
<td>Karanja seed churana</td>
<td>1600</td>
<td>51.06±5.73</td>
<td>40.39</td>
</tr>
</tbody>
</table>

Data: Mean±S.E.M

An apparent suppression of oedema formation was observed at both the dose levels. At 800 mg/kg dose level, 11.81% suppression of oedema was observed and it was statistically non-significant. At higher dose level (1600 mg/ kg), the suppression was 40.39% and it was statistically significant (p< 0.05).

ANALGESIC ACTIVITY
Acetic acid induced writhing [7]
The method employed was that of Koster (1959), which is a modification of phenylquinone writhing syndrome of Siegmund et al (1957), as described by Witkin et al (1961). The dose of the acetic acid (3% v/v) used was 1 ml per 100 g body weight. Acetic acid in aqueous solution in the above mentioned dose produces a writhing syndrome characterized by intermittent contraction of the abdomen, twitching and turning of the trunk and extension of hind limbs, beginning 3-10 minutes after the injection of acetic acid and persisting for 3-4 hours. Only those mice who exhibited this syndrome were used. 18 such albino mice were taken and grouped into three groups after weighing carefully. All the mice were given acetic acid in aqueous solution by intra-peritoneal route at a dose of 1 ml per 100 gm body weight, 45 minutes after test drug administration. Group I and II were pretreated with Karanja seed kwatha (decoction) in a dose of 0.1 ml per 10 g and 0.05 ml per
Peripheral analgesics like aspirin significantly suppress writhing syndrome.

RESULTS AND OBSERVATIONS
Data on effect of Karanja seed decoction (kwatha) on acetic acid induced writhing in mice are shown in Table 2.

Table 2: Effect of Karanja seed kwatha on acetic acid induced writhing mice.

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose extractive material contained in the crude drug</th>
<th>No. of acetic acid writhing within 30 minutes after injection</th>
<th>Percentage of decrease</th>
<th>24 hours mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>-</td>
<td>53.33±14.29</td>
<td>-</td>
<td>6/6</td>
</tr>
<tr>
<td>Karanja seed kwatha</td>
<td>2.5</td>
<td>35.17±13.96</td>
<td>34.06</td>
<td>4/6</td>
</tr>
<tr>
<td>Karanja seed kwatha</td>
<td>5.0</td>
<td>37.17±8.64</td>
<td>30.31</td>
<td>4/6</td>
</tr>
</tbody>
</table>

Data: Mean±S.E.M

In lower test drug dose group (2.5 g/kg), 34.06% decrease in number of acetic acid induced writhing was observed, while in higher test drug dose group (5.0 g/kg) 30.31% decrease was observed. However, both changes were found to be statistically non-significant. Within 24 hours of acetic acid injection all the mice died in control group, whereas even after 24 hours two mice each in lower and higher dose group remained alive. In higher dose group, one mouse remained dull during 30 minutes after acetic acid injection and did not show any writhing.

DISCUSSION

Anti-inflammatory activity
When the Karanja seed churana was evaluated for anti-inflammatory activity in carrageenan induced hind paw oedema in rats, which served both as a primary screening test and representative model of acute inflammation; moderate to significant anti-inflammatory activity was observed at the dose level studied. Karanja seed churana produced 11.81% inhibition at 800mg/kg dose level and 40.39% inhibition at 1600 mg/kg dose level in comparison to control group. Carrageenan oedema represents the exudative phase of acute inflammation, during which there will be an early fluid exudation followed by cell emigration. From the above account, it is clear that inflammation is a multi-factorial phenomenon. It can be speculated that the test drug may have modulatory effect on these factors leading to suppression of inflammation.

ANALGESIC ACTIVITY
The test drug was evaluated for analgesic activity by noting their effect on acetic acid writhing in mice. Expression of the writhing syndrome with acetic acid is reported to be dependant on the continuous formation and release of proligistic mediators especially prostaglandin E, thus it is considered to be an ideal model for assessing peripheral analgesic agents like non-steroidal anti-inflammatory agents. Karanja seed kwatha produced moderate antagonism of acetic acid writhing in mice indicating presence of peripheral analgesic activity. It produced 34.06% suppression at 2.5g/kg dose and 30.31% suppression at 5.0g/kg dose. The analgesics possibly act by raising the threshold of pain stimuli and prevent nerve impulse carrying pain sensation to reach consciousness. This depressant effect is presumably accompanied by some temporary alteration in the biochemical activity of the cells, responsible for the reception of pain sensation in the brain, by means of analgesic drugs. It is possible that the test drug may contain active principles that possess this type of activity.

SUMMARY AND CONCLUSION
Results of the present study clearly show that Karanja (Pongamia pinnata Pierre) seed possess significant anti-inflammatory activity and weak peripheral analgesic activity.

REFERENCES