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EVALUATION OF ANTIULCER ACTIVITY IN *CANNA INDICA* RHIZOMES

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Abstract :
The use of plants as medicines predates written human history. The plant is a biosynthetic laboratory, not only for chemical compounds, but also a multitude of compounds like glycosides, alkaloids etc. These exert physiological and therapeutic effect. Plants can provide biologically active molecules and lead structures for the development of modified derivatives with enhanced activity and reduced toxicity. The small fraction of flowering plants that have so far been investigated have yielded about 120 therapeutic agents of known structure from about 90 species of plants. According to one estimate only 20% of the plant flora have been studied and 60%of synthetic medicines owe their origin to plants. The medicinal values of many plants have been established and published, but most of them are waiting to be explored till date. Therefore, there is every need to find their uses and to conduct pharmacological studies to discover their medicinal properties. Canna Indica (L.) is a spreading annual herb found throughout India but unfortunately it is one of the neglected plants. Hence, the present review aims to open new avenues for the improvement of medicinal use of Canna Indica for various ailments and to bring the anti-ulcer Medicinal plant to the scientists' notice, and raise awareness and add value to the resource. This work attempts to highlight the available literature on Canna Indica (L.) with respect to ethno botany, chemical constituents and summary of various pharmacologic activities.

Key Words: *Canna Indica , Extraction*

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INTRODUCTION:

Herbal products are also commonly used by patients with certain chronic medical conditions including breast cancer (12%), liver disease (21%), human immunodeficiency virus (22%), asthma (24%), and rheumatological disorders (26%). Even as we commence the new century with its exciting prospect of gene therapy, herbal medicines remain as one of the common forms of therapy available to the world population (Rao et al., 1999).

With the rapid progress and advancement in various fields of human activities, the field of medicine and allied sciences have also made rapid strides. The synthesis of many chemicals and their introduction into therapeutics as drugs has certainly revolutionized the treatment of diseases. Today very large numbers of synthetic drugs are available to combat with different diseased conditions.

If one tries to go back to indigenous drugs and bring out the strategies for remedies for ailments people will look down at them since there is a surfeit of established synthetic drugs in modern medicine. The strongest critics are persons who advocate synthetic drugs for this type of studies. However one may not have Jealousy for the pride of place given to synthetic drugs, it is a fact that these drugs are not really away from side effects. They are known to be responsible for large number of adverse reactions and have been the cause of a number of estrogenic diseases. Another consideration for thinking about indigenous practices may be due to the cost factor. Many of the synthetic drugs are abnormally costly beyond the reach of common man. It is natural under these conditions to understand the intention of the researchers in this field of natural products and the drugs obtained from out of the rich flora and fauna of this ancient land and hence appreciable.

Peptic ulcers are very common. If a peptic ulcer is in the stomach it is generally called agastric ulcer whereas a peptic ulcer in the beginning of the small intestines is known as aduodenal ulcer. Most ulcers only harm the first layer of stomach lining and generally heal on their own. They can however progress to the point of being very dangerous.

Symptoms of ulcers may include stomach pain, nausea, fatigue, heartburn, indigestion, chest pain and vomiting or diarrhea. If the bleeding is substantial the vomit and diarrhea will take on a wet coffee grinds appearance due to the digested blood present.

Ulcers can be caused by medications such as Non-steroidal anti-inflammatories (NSAIDs) or cortico-steroids but the most common cause is a bacteria known as H-Pylori. It is important for the doctor to know the cause of the ulcer as the cause dictates treatment.

Many different things may done in order to evaluate the presence of an ulcer. In order to look directly at a suspected ulcer, an endoscopy will often be performed. During this procedure the patient is given sedation medication and then a thin camera is inserted down the esophagus to the stomach and into the small intestines. During the procedure tissue samples will be taken in order to help confirm the presence of H Pylori and to look at the severity of the damage. There are also stool and breath tests that can be done now to look for the presence of H Pylori if an ulcer is suspected and the doctor doesn't feel an endoscopy is necessary. Blood work and stool samples will also be taken to check blood counts/confirm internal bleeding. All of these tests can also be used to rule out other conditions that may mimic the symptoms of an ulcer.

Canna indica was originally named by the eighteenth century Swedish botanist Carl von Linne, usually known by his latinized name of Carolus Linnaeus. Linnaeus apparently thought this species was native to India--hence, the specific epithet of indica. Some older references list this species by the synonym C. coccinea. Another similar species listed for the lovely island of Dominica is C. lambertii, but this name does not appear in most of the more recent botanical references for the Caribbean region. Indian shot has many common names throughout the New World tropics, including canna lily, calenda, English shot, Queensland arrowroot, and the Creole name of "toolima" which is derived from the French "tous les mois." It is also known by the French name "balisier rouge."

The name "Queensland arrowroot" refers to a South American species, Canna edulis, known locally as "achira" and "tous les mois." Achira is grown in the Andes for its starchy, tuberous rhizome which is boiled and eaten. In Peru the rhizomes are baked for up to 12 hours after which they become a white, mucilaginous mass with a sweet taste. At Cuzco the baked rhizomes are sold at the festival of Corpus Christi, and in Colombia they are ground into flour and made into cakes. The easily digested "arrowroot starch" comes from the dried, finely-powdered rhizomes. As its common name implies, C. edulis is also grown in Queensland.

Natural resources also supply basic compounds that may be modified slightly to render them more effective or less toxic. Natural products are used as prototypes or models for synthetic drugs possessing

physiological activities similar to the original drug (Ashutoushkar et al., 2003).

Ulcer is defined as the erosion in the lining of the stomach or duodenum and is caused by the disruptions of the gastric mucosal defense and repair systems. Ulcer in the stomach is called gastric ulcer and in the duodenum is called duodenal ulcer, together peptic ulcer (Gregory et al., 2009).

Peptic ulcer is one of the most common, chronic gastrointestinal disorders in the modern era. Now it has become a common global health problem affecting a large number of people worldwide (Dharmani et al., 2003). In an attempt to search a potent and safe medication for peptic ulcer, Canna indica rhizomes is being screened for its anti ulcer activity.

Plant material:

The leaves of *Canna indica rhizomes* was collected from the hills of tirumala region, chittoor (dist).A.P,India. The plant was identified and auntheticated by Dr.Madhavachetty, Assisatant professor, Departmant of botony, S.V. University, Tirupathi. The rhizomesof the plant were stored in herbarium at the college for further reference.

Preparation of extract:

The rhizomes of *Canna indica*(100 g) was extracted by methanol (60-80º C) for 6 hours in Soxhlet extractor. The methanol extract was evaporated in vacuum until a constant weight was achieved. MECI means methanol extract of *Canna indica*. It gave 2.44% of the residue.

Animal:

Albino rats (150-200g) of either sex were used for the experiment. They were kept in the animal house in a controlled room temperature at 25+2 c, relative humidity 44-56%, light and dark cycles of 10 and 14 hr, respectively for 1 week before the experiment. The animals were grouped and housed in polyacrylic cages for the further experiment.

Drugs and Chemical:

Extract: The MECI was dissolved in 1 % tween 80 as a vehicle and administered P.O in a dose of 100 mg/ kg, 200 mg/ kg and 400 mg/kg.

Standard drug: The ranitidine was dissolved in 1% tween 80 and administered i.p in a dose of 27mg/kg.

Acute Toxicity studies:

The Acute Toxicity studies were performed in order to establish the therapeutic index of a test drug. The experiment was conducted according to the OECD, 423 guidelines. It was administered as 5,100, 1000 and 2000 mg/kg.

Experimental design:

Rats were divided in to 5 groups. Each group contains 6 rats and treated with the following drug for five successive days and 6th day aspirin was administered and to evaluate the antiulcer activity.

Group 1: Control - Normal saline (1ml/kg)
Group 2: Ranitidine - 27mg/kg (standard control)
Group 3: MECI - 100 mg/kg
Group 4: MECI - 200mg/kg
Group 5: MECI - 400mg/kg.

ASPIRIN INDUCED ULCER METHOD:

Aspirin at a dose of 200mg/kg (20mg/ml suspension in 1%CMC (Carboxy methyl cellulose)) was administered orally to 18hr fasted animals. After 1hr the test drug was administered on 6th day. The ulcers were scored after 4hrs. The stomach was taken out and cut open along the greater curvature and ulcers were scored by a person in the glandular portion of the stomach. The ulcer index was calculated by adding the total number of ulcers per stomach and the total severity of ulcer per stomach *(Gerhard et al., 2002)*.

PYLORUS LIGATION INDUCED ULCER METHOD:

This is the most acceptale method *(Gregory et al., 2009)*. Rats were divided in to 5 groups. Each group contains 6 rats. Each weighing about 150-200gm and fasted for 4hr with free access to water. Pylorus ligation induced uler method was performed under diethyl ether anaesthesia to each animal. Animals were given to MECI 100 mg/kg, 200 mg/kg and 400 mg/kg b.w. ranitidine was prepared in 1% tween 80 suspension as a vehicle orally immediately after pylorus ligation.

Animals were sacrified 6hrs later the stomach was carefully removed and gastric contents were collected. The gastric juice was centrifused at 3000 rpm for 30 mins and then volume of gastric juice was measured. Acidity in the supernant was determined by titration with0.01N NaOH and expressed as m.eq/l. the stomach was cut open along the greature curvature and pinned on a soft board for evaluating gastric ulcers and ulcer index is calculated using formula:

Ulcer index = 10/x

Where x = Total mucosal area / Total ulcerated area.

RESULTS AND DISCUSSION:

The etiology of peptic ulcer is unknown in most of the cases, yet it is generally accepted that it results from an imbalance between aggressive factors and the maintenance of mucosal integrity through the endogenous defence mechanisms. To regain the balance, different therapeutic agents including plant extracts may be used *(Raju et al., 2009)*.

Various factors that have been implicated in the pathogenesis of gastric ulcers are an increase in gastric acid secretion, pepsin activity and oxidative stress in the gastric mucosa, and a decrease in mucous and bicarbonate secretion *(Wallace et al., 1996), (Granger et al., 1986).*

ASPRIN – INDUCED GASTRIC ULCER METHOD:

Groups	ASPIRIN (mg/kg)	Ulcer Index	Ulcer Inhibition (%)
Ulcer Control0.9 % saline	-	3.981± 0.5631	-
Ranitidine(27 mg/kg)	0.2	1.16+ 0.1667 **	48.32
MECI(100 mg/kg)	0.2	2.213±0.987**	19.35
MECI(200 mg/kg)	0.2	2.85± 0.1763**	23.61
MECI(400 mg/kg)	0.2	3.652± 0.8456***	43.89

N= 6 animals in each group.

Table 1: Values are expressed in Mean + SEM, (n=6), when compared with control, P<0.05, P<0.01, P<0.001 *, **, *** respectively, Oneway ANOVA followed by Dunnet's t – Test.

NSAIDs like indomethacin and aspirin induces gastric lesions by inhibiting cyclo-oxygenase (COX) resulting in less formation of prostacyclin, the predominant prostanoid produced in the gastric mucosa.

The general ulcer inducing methods are aspirin induced ulcer, pyrolic ligation method, cold restraint stress induced ulcers, ethanol induced ulcers, cold water immersion method, swimming stress ulcer method. These models represent some of the most common causes of gastric ulcer in humans.

Ulcer is defined as the erosion in the lining of the stomach or duodenum and is caused by the disruptions of the gastric mucosal defense and repair systems. Ulcer in the stomach is called gastric ulcer and in the duodenum is called duodenal ulcer, together peptic ulcer

Group 2: Ranitidine - 27mg/kg (standard control)

Group 3: Methanol extracts of *Canna indica rhizomes* - 100 mg/kg

Group 4: Methanol extracts of *Canna indica rhizomes* - 200mg/kg

Group 5: Methanol extracts of *Canna indica rhizomes* - 400mg/kg.

Here we are represents the ulcer inhibition percentage in folloing table 1. The standard drug of ranitidine showed 48.32 % of ulcer inhibition. The % inhibition of (Figure 1.) 100 mg/kg, 200 mg/kg, and 400 mg/kg values are 19.35, 23.61, and 43.89 respectively. If the concentration of MECI increases ulcer percentage inhibition also increase. So this is having dose dependent manner.

PYLORUS LIGATED INDUCED ULCER METHOD:

The causes of gastric ulcer pyloric ligation are believed to be due to stress induced increase in gastric hydrochloric acid secretion and/or stasis of acid and the volume of secretion is also an important factor in the formation of ulcer due to exposure of the unprotected lumen of the stomach to the accumulating acid *(Raju et al., 2009).*

Pylorus ligation induced ulcers are due to auto digestion of the gastric mucosa and breakdown of the gastric mucosal barrier. These factors are associated with the development of upper gastrointestinal damage including lesions, ulcers and life threatening perforation and hemorrhage. Aspirin, phenylbutazone, indomethacin and some non-steroidal anti-inflammatory drugs are also known to cause duodenal and gastric ulceration. Prostaglandin E2 and I2 are predominantly synthesized by the gastric mucosa and are known to inhibit the secretion of gastric acid and stimulate the secretion of mucus and bicarbonate.

Hydrophobic surfactant-like phospholipids secretion in the gastric epithelial cells is also stimulated by the prostaglandin. It is also showed development of gastric ulcers in pyloric ligation model. Volume of gastric secretion is an important factor in the production of ulcer due to exposure of unprotected lumen of the stomach to the accumulating acid *(Raju et al., 2009).*

The anti ulcer activity of the plant of Methanol extract of *Canna indica* rhizomes was evaluated by employing pyloric ligation induced ulcer method & aspirin induced ulcer methods. Ulcers results from extrinsic factors like Helicobacter pylori, Non steroidal anti-inflammatory drugs (NSAIDs), Tobacco smoking, Alcohol, Stress and Intrinsic factors like Acid & Pepsin, Acid Reflux. These factors can be used to induce ulcer in animal models.

Groups	Dose (mg/kg)	Gastric volume (ml)	P^H	Free Acidity
Ulcer Control	0.9 % saline	2.84±0.5978	2.85±0.0916	56.83±0.833
Ranitidine	(27 mg/kg)	3.91±0.1128 **	4.53±0.0954 **	35.16±1.014 **
MECI	(100 mg/kg)	2.127± 0.784**	2.513±0.8087**	4.85±0.894*
MECI	(200 mg/kg)	2.997±0.482**	3.68±0.8061 **	41.5±0.738 **
MECI	(400 mg/kg)	3.590±0.436***	3.75±0.2565 **	37.16±0.303 **

N= 6 animals in each group.

MECI: Methanol extracts of *Canna indica* rhizomes.

Table 2: Values are expressed in Mean + SEM, (n=6), when compared with control, P<0.05, P<0.01, P<0.001, *, **, *** respectively, Oneway ANOVA followed by Dunnet's t – Test. Gastric volumes were significant to tested groups. Ranitidine showed 3. 91, methanolic extracts of *Canna indic rhizomes* 100 mg/kg, 200 mg/kg and 400 mg/kg, had 2.127, 2.997, 3.59 respectively. So extracts represents dose dependent.

Here we are represents the ulcer inhibition percentage in folloing table 3, figure 2. The standard drug of ranitidine showed 79.23 % of ulcer inhibition. The % inhibition of 100 mg/kg, 200 mg/kg, and 400 mg/kg values are 28.3, 53.46, and 65.45 respectively. If the concentration of MECI increases ulcer percentage inhibition also increase. So rhizomes of *Canna indica*showed dose dependent manner.

Groups	Dose (mg/kg)	Ulcer Index	Ulcer Inhibition (%)
Ulcer Control	0.9 % saline	5.58+ 0.2126	-
Ranitidine	(27 mg/kg)	1.937+0.1675 **	79.23
MECI	(100 mg/kg)	2.013±0.987**	28.3
MECI	(200 mg/kg)	2.95+ 0.4641 **	53.46
MECI	(400 mg/kg)	3.58+0.1257 **	65.45

N= 6 animals in each group.

Table 3: Values are expressed in Mean + SEM, (n=6), when compared with control,P<0.05, P<0.01, P<0.001 *, **, *** respectively, Oneway ANOVA followed by Dunnet's t – Test.

Group 2: Ranitidine - 27mg/kg (standard control)

Group 3: Methanol extracts of *Canna indica rhizomes* - 100 mg/kg

Group 4: Methanol extracts of *Canna indica rhizomes* - 200mg/kg

Group 5: Methanol extracts of *Canna indica rhizomes* - 400mg/kg.

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From the above results, we observed that Methanol extract of *Canna indica* rhizomes significant anti ulcer activity by pyloric ligation induced method & aspirin induced methods againstgastric ulcers in rats.

CONCLUSION:

In the present study, methanolic extract of *Canna indica rhizomes* showed a promising antiulcer effect on peptic ulcer models in pyloric ligation and aspirin induced methods in rats. Hence oral administration of the methanolic extract of *Canna indica rhizomes* have the potential to reduce the formation of peptic ulcers.

This property of methanolic extract of *Canna indica rhizomes* act would be highly beneficial for treatment of various types of peptic ulcers in human beings and reduce the cost burden of the society. However further the active principles of *Canna indica rhizomes* responsible for this property is too isolated phytochemically and studies with these purified constituents on different ulcer models are waiting to understand the complete mechanism of antiulcer activity.

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