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EFFECT OF RICINUS COMMUNIS SEED OIL (CASTOR OIL) ON EXPERIMENTAL (ETHANOL-INDUCED) GASTRIC ULCER IN RATS.

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ABSTRACT

Ricinus communis seed oil (castor oil) is used to relieve some gastrointestinal symptoms and as a bowel evacuate as a preliminary to intestinal examination. The study was aimed at the investigation of the effects of castor oil on ethanol induced gastric ulcer model in rats. Pretreatment of rats orally with castor oil (1 ml/rat) significantly ($p<0.05$) protected the rats (60%) from ulcers produced by 99% ethanol, with 1.7 ± 0.6 mm ulcer index compared to the control (4.9 ± 0.3mm). Similar pretreatment with Cimetidine (200mg Kg^{-1}) protected the rats only by 40% with 2.8 ± 0.5mm ulcer index. Gastric ulcers in rats initially challenged with 99% ethanol were inhibited by 60%, 40% and 20% with corresponding ulcer index of 2.6 ± 0.5mm, 3.1 ± 0.2mm and 3.6 ± 0.8mm by Castor oil, Cimetidine, and Omeprazole (20mgkg^{-1} p.o) respectively, compared to the control (4.6 ± 0.4mm). The result show that castor oil has both preventive effects and curative effect in ethanol – induced gastric ulcer.

KEY WORDS: Ethanol, Gastric ulcer, Castor oil, Cimetidine and Omeprazole.

INTRODUCTION

The castor oil plant Ricinus communis (Family Euphorbiacceae) is indigenous to India. The principal producing Countries include India, Brazil, the then Soviet Union and Thailand (Evans, 1989). Peasant farmers of Nigeria and Cameroon also cultivate it. The seeds are oval and slightly compressed, with considerable differences in size and colour. The seeds in good condition have a very little odour, taste and acrid. Ricinus communis seed oil (Castor oil) is a colourless or a pale yellow liquid with a slight odour and faintly acrid taste (Evans, 1989). The oil contains fixed oil which consists of the glycerides of ricinoleic, isoricinoleic, stearic and dihydroxystearic acids (Wallis, 1985). The use of castor oil to ease constipation, induce vomiting or as a laxative has been documented (Bertram 1989, Goodman and Gilman, 1991, and Elizabeth et al., 1996). It is equally used clinically as bowel evacuate after food poisoning as a preliminary to intestinal examination (Bertram, 1989). In most rural areas of Nigeria and Cameroon, castor oil is taken in the early hours of the day to produce purgative effect at the dawn of the day. Since caster oil has a high viscosity (Martin, 1981). It is likely that it may protect the gastric mucosal barrier and so, may be beneficial in the treatment of peptic ulceration. The investigation was therefore aimed at the effects of Ricinus communis seed oil (castor oil) on ethanol-induced gatric ulcer in rats.

MATERIALS AND METHODS

EXPERIMENTAL ANIMALS

Wistar albino rats of both sexes (100 – 175g) from the Animal House, Department of Pharmacology & Toxicology, Faculty of Pharmacy, University of Uyo, Nigeria were used. The animals were housed in plastic cages with stainless steel cages for adequate ventilation. The were fed with grower's mash feed (Akwa Feed, Uyo, Nigeria) and had free access to water under standard condition of illumination (12h dark - 12h light), humidity (60 – 100%) and temperature (28 ± 2° c). For the tests, animals were weighed and randomly allocated into groups of five rats per group. Food was withdrawn 14 – 16hr before all experiments because the standard drugs, castor oil or the ulcerogenic agent were administered orally.

DRUGS

Cimetidine (Smith Kline and French Labs. England) and Omeprazole (V.S. Interantional P.V.T India) were used as the standard reference

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drugs. All reagents were of analytical grade except otherwise indicated. The substances and reagents were prepared immediately before use.

PREPARATION OF SEEDS AND EXTRACTION OF CASTOR OIL.

Mature and dry Ricimus communis seeds (castor seeds) were harvested in the Month of December, 2000 from the medicinal plant farm of the Department of Pharmacognosy, Faculty of Pharmacy, University of Uyo, Nigeria . The seeds were identified and authenticated by U.A. Essiett of the Department of Botany, University of Uyo. Herbarium Specimens (HS/068/2000) are deposited in the Department of Pharmacognosy Hebarium. The seeds were further sun dried in open air and the shells removed. After removing the tasta, the seeds were macerated into a paste. The castor oil was extracted by an improvised hot water method. In brief, the paste was dissolved in cool water which was boiled with continuous stirring. When fully boiled it was allowed to stand until oil coalesced at the surface. The oil which contained both residue of macerated seeds and some traces of water was carefully collected into a clean clay pot and was again boiled to eliminate the traces of water. The residue of the macerated seeds adhered to the bottom of the pot, leaving a characteristic pale yellow castor oil. After cooling, the oil was carefully filtered into clean conical flask, corked and stored until required. The process was repeated to obtain enough castor oil for the investigation.

ANTI- ULCEROGENIC ACTIVITIES.

Ethanol-Induced Ulcer

The ethanol-induced ulcer assay was carried out in rats according to the method of Morimoto et al., (1991). Rats were weighted and randomly divided into groups of five rats per group. Two sets of experiments were carried out. The rats were fasted for 14 –16 hrs, with free access to water before each experiment.

In the first set of experiments, group 1 rats (control) were given the vehicle, distilled water (1ml, p.o), while groups II and III were given Cimetidine (200mg kg^{-1}, p.o) and Castor oil (1ml, p.o) respectively. One milliliters of 99% ethanol to induce ulcer was then given to the rats orally, which 1hr, previously had been treated either with distilled water, Cimetidine or castor oil. The rats were killed with chloroform 1hr later.

In the second set of experiments, one millilitre of 99% ethanol was given by gavage to each of the four groups of rats to induce ulcer. Group II, III and IV were 1 hr later given orally Cimetidine (200Mg kg^{-1}), omeprazole (20mg kg^{-1}), and Castor oil (1ml/rat) respectively whereas group I received nothing except the original 99% ethanol. The rats were killed 1hr later with chloroform. In both sets of experiments, the stomachs were excised and inflated by injection of normal saline (2ml). The stomachs were fixed by injection of 5% formalin for 30min and opened along the greater curavature.

The stomachs were carefully cleaned out and examined for ulceration with the aid of a hand lens. The extension of the ulcer lesion was measured according to the method of Merazzi-Ubertic and Turba as described by Giodano et al., (1990) and scored as follows:

GRADE	DESCRIPTION
0	**No ulcer**
1	Haemorrhagic and slightly dispersed ulcer less than 2mm in length
2	One ulcer as above, upto 5mm in length
3	More than one ulcer grade 2
4	One ulcer above, 5mm in length
5	More than one grade 4 ulcer

The results were expressed in terms of an ulcer index (UI) which is the average severity of erosion/rat for each group on the scale from o to 5. The number of animals divided the sum of these values. The percentage inhibition of the ulcer was calculated based on the number of rats in each group with no discernible ulceration.

DATA ANALYSIS

Data were expressed as means ± standard deviation and analyzed by the use of student's t-test. $P<0.05$ was regarded as indicative of significance.

RESULTS AND DISCUSSION

The prevention of ethanol induced gastric ulcer with the pretreatment with either Cimetidine or Castor oil was studied in rats and the results are shown in Table 1.99% ethanol (1ml/rat) given to rats by oral gavage produced the clearly expected characteristic zonal necrotic mucosa; lesions to the control group. Castor oil (1Ml/rat) was given orally while Cimetidine

(200mgkg^{-1}) was also given by the same route. The ulcer index for the control group was 4.9 ± 0.6mm, Cimetidine pretreated rats (2.8 ± 0.5mm) and 1.7 ± 0.6mm for Castor oil pretreated rats. The percentage gastric ulcer prevention were 0,40 and 60 for control, Cimetidine and Castor oil respectively.

The percentage gastric ulcer prevention was obtained based on the number of rats per group in which ulcerations were not observed. Both the anti-ulcer drug, Cimetidine and Castor oil significantly inhibited ulcer formation ($P<0.05$) compared to the control. Pretreatment of rats with either Cimetidine or castor oil indicated that castor oil was more effective than Cimetidine in the prevention of ethanol-induced ulcers at the given doses.

The effects of oral administration of either Castor oil (1ml/rat), Cimetidine (200mg kg^{-1}) or Omeprazole (20mg kg^{-1}) after the induction of ulcer with 99% ethanol in rats is shown in Table 2. Similarly, the result indicated

TABLE 1 : Effect of Pretreatment of rats with either Castor oil or Cimetidine on ethanol – induced gastric ulcer in rats.

Group	Treatment	Dose	No. of rats	No. of stomachs per score	Ulcer prevention (%)	Ulcer index (mm)
I	Ethanol only	1ml/rat	5	2/4 3/5	0	4.9 ± 0.3
II	Cimetidine + Ethanol	200mg kg^{-1}	5	2/0 1/1 2/3	40	* 2.8 + 0.5
III	Castor Oil + ethanol	1ml/rat	5	3/0 1/1 1/2	60	* 1.7 ± 0.6

Rats were given 99% ethanol one hour after oral administration of either Castor oil or Cimetidine. They were killed 1 hour later with chloroform. Results are expressed as MEAN ± standard deviation and were analysed using the student's t-test: *P< 0.05 compared with the results from only ethanol treatment (control).

TABLE 2: Effects of Cimetidine, Omeprazole or Castor oil ethanol – induced gastric ulcers in rats.

Group	Treatment	Dose	No. of rats	No. of stomachs per score	Ulcer inhibition (%)	Ulcer index (mm)
I	Ethanol only	1ml/rat	5	1/2 2/3 1/4 1/5	0	4.6 ± 0.4
II	Ethanol + Cimetidine	200mg kg^{-1}	5	1/1 2/2 2/0	40	* 3.1 ± 0.2
III	Ethanol + Omeprazole	20mg kg^{-1}	5	2/3 1/4 1/5 1/0	20	* 3.6 ± 0.8
IV	Ethanol + Castor oil	1ml/rat	5	1/1 1/2 3/0	60	* 2.6 ± 0.5

Rats were given 99% ethanol orally, 1hr later they were administered either Cimetidine, Omeprazole or Castor oil by the same route. They were killed 1 hour later with chloroform. Results are expressed as MEAN ± standard deviation and were analysed using the student's t-test: * P<0.05 compared with the results from only ethanol treatment (control).

a clearly characteristic zonal necrotic mucosal lesions in the control (4.6 ± 0.4mm).

The results indicated that Cimetidine, Omeprazole and Castor oil inhibited ulcers by 40%, 20% and 60% respectively, with ulcer index of 3.1 ± 0.2mm, 3.6 ± 0.8mm and 2.6 ± 0.5mm respectively. The percentage gastric ulcer inhibition was based on the number of rats per group with no discernible ulcerations. Castor oil significantly inhited ($P<0.05$) ethanol – induced gastric ulcer compared to the control. The results again indicated that castor oil is more effective than Cimetidine and Omeprazole in ethanol challenged ulcers followed by treatment with either Castor oil, Cimetidine or Omeprazole. Szabo (1987) reported that ethanol treatment induces solubilization of mucus constituents in the stomach with a concomitant fall in the transmucosal potential difference, and increases NA^+ and K^+ flux into the lumen, pepsin secretion, the loss of H^+ and the histamine content in the lumen as well as depresses tissue levels of DNA, RNA and proteins leading to flow stasis in injured areas. In the present study, Castor oil significantly produced protective effects against gastric mucosal injury induced by ethanol. The observed effects of Castor oil could be attributed to the fact that Castor oil has an extremely high viscosity (Martin, 1981). Hence, the castor oil possibly acted as a mucosal protectant and protected the stomach of the rats from the ethanol- induced ulceration. Omeprazole is a substituted benzimidazole that inhibits the gastric parietal cells proton pump (H^+, K^+ ATPase). It requires activation in the acid environment of the secretory canalicus of the parietal cells (Wallmark, 1986). Its observed low anti-ulcer effect (20%) on the ethanol-induced ulcer in this study could be attributed to the fact that the ulcer induction was possibly not through gastric acid secretory process . Similarly, Cimetidine is an Histamine H^2 – receptor antagonist. Its relatively low activity compared to Castor oil in the study could be attributed to the possibility that histamine release may not be involved. The present study was a pilot study undertaken with the view of establishing the possible effects of Castor oil in individuals who may have gastric ulcer with a view to treating themselves with castor oil. Also, since oral administration of castor oil produced ulcer protective effects in the ethanol –induced ulcers studied in rats, regular castor oil administration may be beneficial to ulcer prone individuals if these results are applicable to man.

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