

EVALUATION OF ANTI -ULCER ACTIVITY OF *Momordica Charantia* IN RATS

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ABSTRACT

Momordica charantia (Cucurbitaceae) is a plant, reported for its variety of ethnic medicinal uses. Hence we have planned to screen antiulcer activity of fruit of the plant with the alcoholic and aqueous extracts. Fruit powder successively extracted with alcohol and water were subjected for phytochemical screening to identify different phytoconstituents. LD_{50} studies for both (alcoholic and aqueous) extracts were conducted upto the dose level of 2 g /kg by following OECD up and down method of guidelines No.425. Anti ulcer activity was evaluated in various animal models like Pylorus ligation, aspirin, Stress induced ulcer models in rats. Preliminary phytochemical studies revealed the presence of saponins, sterols, mucilage, glycosides, alkaloids, steroidal saponins in both the alcoholic and aqueous extracts of *M.charantia*. No mortality was observed with any of the 2 extracts up to the maximum dose level of 2 g/kg. Further alcoholic and aqueous extracts at 200 and 400 mg/kg, p.o but not with 100 mg/kg p.o doses significantly ($P < 0.01$) reduced the ulcer score, ulcer number, ulcer index, free acidity and total acidity in Pylorus ligation, aspirin, Stress induced ulcer models in rats. The present study revealed the antiulcer activity of fruit extracts of *M.charantia* and the activities are due to the presence of phytochemical constituents^{1,2,3} such as saponins, sterols, mucilage, glycoside, alkaloids, steroidal saponins as these phytochemical constituents were already reported for the above mentioned effects.

KEYWORDS: *M. charantia*, fruit, pylorus ligation, stress, aspirin, Anti ulcer activity.

INTRODUCTION:

Gastric ulcers the most wide state disease and are a very common global problem today. Peptic ulcer is a lesion of the gastric /duodenal mucosa occurs at a site where the mucosal epithelium is exposed to acid and pepsin. Peptic ulcers occurs due to imbalance between the offensive (gastric acid secretion) and defensive (gastric mucosal integrity) factors. The aggressive and protective factors in the stomach are acid pepsin secretion, mucosal barrier, blood flow, cellular regeneration,

prostaglandins and epidermal growth factors. Sometimes the gastric mucosa is continuously exposed to potentially injurious agents such as pepsin, bile acids, food ingredients, bacterial products and drugs. Factors such as stress, smoking, nutritional deficiency and ingestion of NSAID'S all can increase the incidence of gastric ulcers. It is reported that prolonged anxiety, emotional stress, haemorrhagic surgical shock, burns and trauma are known to cause severe gastric irritation.

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Physical, chemical and psychological factors may lead to gastric ulceration in humans and experimental animals. Reactive oxygen species (ROS) are reported in the pathophysiology of human diseases such as neurodegenerative inflammation, viral infections, autoimmune GI inflammation and gastric ulcer.

The current medicinal treatment of peptic ulcer aims for

1. Inhibition of gastric acid secretion
2. Reinforcement of gastric mucosal production

The currently used antiulcer drugs like H_2 –receptors blockers, proton pump inhibitors, antimuscarinics produce adverse reactions such as hypersensitivity, arrhythmia, impotence and haemopoietic changes with a possibility of increased rate of ulcer recurrence within one year after cessation of the treatment.

Because of the above mentioned demerits reported with the current antiulcer therapy there is a need for the search of newer therapeutic antiulcer agents from plant sources from the alternative therapy Ayurvedha.

Plant extracts some of the most attractive sources of new drugs show to produce promising and favourable reasons in the treatment of gastric ulcer further in the traditional medicine ayurvedha, several plants and herbs are advocated for the treatment of gastrointestinal disorders including gastric ulcers.

No scientific data is available in support of traditional uses of many plants including anti-arthritis and anti-inflammatory activity of *M.charantia*.

Hence the present study was planned to evaluate antiulcer activity of alcoholic and aqueous extracts of fruits of *M.charantia* in pylorus ligation, stress and aspirin induced gastric ulcer model in experimental animals, rats.

In pylorus ligation induced ulcer model gastric ulcers are due to over production of gastric acid or decrease in gastric mucous production. In this model an increase in acid-pepsin accumulation due to pylorus obstruction and subsequent digestion of the mucous was reported. The cause of gastric ulcers after pylorus ligation are due to an increase in gastric HCl secretion, stasis of acid due to stress and over production of gastric acid increased volume is also an important factor involved in ulcer formation as the unprotected lumen of the stomach is exposed to the accumulated acid.

In this model both alcoholic and aqueous extracts of fruits of *M.charantia* have significantly reduced the number of ulcers, ulcer index, gastric volume, free and total acids. Antiulcer activity of alcoholic extract was recorded relatively better than the aqueous extract. The difference in antiulcer activity can be accounted for the number and quantity of the phytoconstituents present with the both the extracts. Phytochemical examination revealed that alcoholic extract answers positively with saponins, Sterols and flavonoids where in aqueous extract noted with flavonoids and saponins. Sterols present in alcoholic extract can be counted for the relatively better antiulcer activity of alcoholic extract in pylorus induced ulcer model, as saponins, sterols and flavonoids are already reported for their antiulcer activity.

It was reported that stress plays an important role in the etiology of gastroduodenal ulcers. Stress induced ulcer is probably mediated by release of histamine which in turn increases gastric secretion and causes disturbance of gastromucosal macrocirculation, alteration in motility and reduced production of mucus. Vagal activity has been suggested as the main factor in stress induced ulceration, as it stimulates HCl in the stomach through the action of Ach acting through muscarinic receptors. During stress vagal activity was upregulated, resulting with the production of more acid.

Further stress also causes mast cell degranulation and decreased synthesis of prostaglandins and complex neurochemical mechanisms are also involved like changes in the synthesis, action and degradation of hormones, neurotransmitters and neuromodulators. CNS also plays an important role in ulceration and regulation of plasma corticosterone.

It was reported that cold restraint stress aggravates the severity of ulcers, lipid peroxidation and plasma corticosterone. Free radicals affect lipids by initiating peroxidation, Superoxide, hydrogen peroxide and hydroxyl radical are important. Reactive oxygen species (ROS) responsible for tissue damage. The higher lipid peroxidation and SOD levels are indication of the increased production of superoxide within the tissue, the restraint effect cell degeneration through lipid peroxidation of membrane lipids, breaking of DNA strands and denaturation of cellular proteins.

Both alcoholic and aqueous extracts of fruit of *M.charantia* have significantly reduced the number of ulcers, gastric volume, free and total

acids. Antiulcer activity of alcoholic extract was noted relatively more than aqueous extract. As mentioned in the above pylorus ligation induced ulcer model the difference in antiulcer activity recorded with the two extracts can be accounted with difference in phytoconstituents and quantity of the same. Presence of sterols only in alcoholic extract can be considered for the better antiulcer activity.

NSAID'S are reported with gastroduodenal ulceration because of their effect on prostaglandin synthesis (PGE and PGI₂) as prostaglandin protects the mucosal layer of stomach from the corrosive effect of gastric acid.

In the stomach prostaglandins play a protective role such as stimulation of HCO₃ ions secretion, maintenance of mucosal blood flow and regulation of mucosal cell turnover and repair. Suppression of prostaglandin synthesis results with the increased susceptibility of mucosal injury and gastroduodenal ulceration. Prostaglandins relax the circular muscle of gastric mucosa and cause flattening of the folds, increasing the mucosal area exposure to necrotic agents and reduce volume of gastric irritants on rostral crest. It was reported that decreased gastric motility exerts a gastroprotective action.

In the present study NSAID'S i.e, aspirin induced gastric ulcer model both the alcoholic and aqueous extracts of fruits of *M.charantia* significantly reduced the number of ulcers, ulcer index, gastric volume, free and total acids.

In this model also alcoholic extract of fruit of *M.charantia* has exhibited relatively better antiulcer activity than the aqueous extract as mentioned earlier. The anti-ulcer effect is increase in are

attributed to the phytoconstituents sterols present extra in alcoholic extract in addition to flavonoids, saponins, mucilage in both the extracts.

Methods and Materials

Anti-ulcer Activity

Pylorus ligation induced ulcer model

The ulcer protective effect of AEFMC and AQEFMC were studied as per the method of Shay et al 1945. The accumulation of acidic gastric juice in the stomach causes ulceration and in this method several parameters were estimated.

Method

Pylorus Ligation Model^{4,5,6} (3 days study)

Albino rats weighing between (160-200 gm) were divided into 8 groups of 6 rats in each. They were fasted in individual cages with measures taken to avoid coprophagy for 24 h prior to the experiment with free access to water. Group A was served as normal control given with vehicle only. Group B with standard drug, groups C, D, E and F, G, H were treated with low, medium and high doses of AEFMC and AQEFMC respectively. The various groups were treated with vehicle/drug/ extracts 30 min prior to pylorus ligation and the details of the protocol was given below: Group A: Normal animals treated with vehicle only; Group B: Standard Ranitidine (10 mg/kg i.p); Group D: Low dose of AEFMC (100 mg/kg); Group E: Medium dose of AEFMC (200 mg/kg); Group F: High dose of AEFMC (400 mg/kg); Group G: Low dose of AQEFMC (100 mg/kg); Group H: Medium dose of AQEFMC (200 mg/kg); Group I: High dose of AQEFMC (400 mg/kg)

EXPERIMENTAL PROCEDURE:

Under light ether anesthesia, the abdomen was opened and the pylorus ligation performed and then sutured. 4 h after pylorus ligation all the animals were sacrificed with excess of anaesthetic ether and the stomach of each rat was dissected out. Gastric juice collected into centrifuge tubes was centrifuged at 1000 rpm for 10 min and volume was noted. The p^H of the gastric juice was recorded by p^H meter. The gastric content was subjected for analysis of free and total acidity. The stomachs were washed under running tap water and then focused under microscope to note the ulcers in the glandular portion. The number of ulcers per stomach were scored microscopically with the help of (10x) hand lens and the scoring is done as per standard procedure. Mean ulcer score for each animal is expressed as Ulcer Index. The percentage ulcer protection was calculated using the formula .

Percentage ulcer protection = $U_t / U_c \times 100$
Where U_t = Ulcer index of treated group and

U_c = Ulcer index of the control group

Table:Ulcer scores

S. No.	Stomach colour	Ulcer score
1	Normal colour	0
2	Red colour	0.5
3	Red spots	1
4	Hemorrhagic streaks	1.5
5	3 > 5 ulcers	2
6	< 5 ulcers	3

Reagents for biochemical estimations of free and total acidity

1) Reagents for estimation of free and total acidity

- Freshly prepared 0.01N oxalic acid solution (BDH) was used to standardize sodium hydroxide.

- b. Freshly prepared 0.01N sodium hydroxide
- c. Topfer's reagent. It is dimethylamino azobenzene 0.5% in absolute ethanol available in 100 ml package.
- d. Freshly prepared 1% Phenolphthalein (BDH) solution prepared in 50% absolute ethanol.

Methods for biochemical estimation of free and total acidity.

Collection of gastric juice

Gastric content collected from pylorus ligated rats was centrifuged and the volume of gastric juice as well as p^H of gastric juice were noted. The gastric juice was subjected to biochemical estimations as follows:

Determination of free and total acidity

1 ml of gastric juice was pipetted into a 100 ml conical flask, 2 or 3 drops of Topfer's reagent was added and titrated with 0.01N Sodium hydroxide until all traces of red colour disappears and the colour of the solution turns to yellowish orange. The volume of alkali added was noted. This volume corresponds to free acidity. Then 2 or 3 drops of phenolphthalein solution was added and titration was continued until a definite red tinge appears. Again the total volume of alkali added was noted now this volume corresponds to total acidity.

Acidity was calculated by using the formula

$$\text{Acidity} = \frac{\text{Volume of NaOH} \times \text{Normality of NaOH} \times 100}{0.1} \text{ meq /lt/ 100g}$$

Cold Stress induced ulcers (cold water immersion method)^{7,8}:

Albino rats weighing between (150-200 g) and each group containing 6 animals were divided into 8 groups.

Group A: Normal animals treated with vehicle only; Group B: Standard Ranitidine (10 mg/kg i.p); Group D: Low dose of AEFMC (100 mg/kg); Group E: Medium dose of AEFMC (200 mg/kg); Group F: High dose of AEFMC (400 mg/kg); Group G: Low dose of AQEFMC (100 mg/kg); Group H: Medium dose of AQEFMC (200 mg/kg); Group I: High dose of AQEFMC (400 mg/kg)

Experimental Procedure:

Albino rats of either sex weighing between (150-200 g) were divided into 8 groups of 6 rats in each. Group A was served as normal control, given with vehicle only. Group B with standard drug. Groups C, D, E and F, G, H treated with low, medium and high doses of AEFMC and AQEFMC respectively. After 30 min of oral administration of the vehicle/standard/extracts rats are placed in cold water vertically for 1h in individual restraint cages maintained at 22°C. Then, they were taken out, dried and injected with 30 mg/kg Evans blue i.v via the tail vein. 10 min later, sacrificed with ether and stomachs are removed. Formol-saline (2%v/v) is then injected into the totally ligated stomachs for overnight storage. The next day, the stomachs opened along the greater curvature, were washed in warm water, and examined microscopically for ulcers with the help of hand lens (10x). Mean ulcer score for each animal is expressed as ulcer index. Gastric juice collected into centrifuge tubes and centrifuged at 1000 rpm for 10 min and the volume was noted. The p^H of the gastric juice was recorded by p^H meter and the gastric content is subjected for analysis of free and total acidity.

Aspirin induced gastric ulcers^{7,9}:

Albino rats of either sex weighing between (150-200 g) each group containing 6 animals were divided into 8 groups. Group A: Normal animals treated with vehicle only; Group B: Standard Ranitidine (10 mg/kg i.p); Group D: Low dose of AEFMC (100 mg/kg); Group E: Medium dose of AEFMC (200 mg/kg); Group F: High dose of AEFMC (400 mg/kg); Group G: Low dose of AQEFMC (100 mg/kg); Group H: Medium dose of AQEFMC (200 mg/kg); Group I: High dose of AQEFMC (400 mg/kg)

Experimental Procedure:

Albino rats of either sex weighing between (160-200 g) were divided into 8 groups of 6 rats in each. Group A was served as normal control given with vehicle only. Group B with standard drug, and groups C, D, E and F, G, H were treated with low, medium and high doses of AEFMC and AQEFMC. Respectively. After 30 min aspirin was administered at a dose of 250 mg/kg p.o, and after 6 h rats were sacrificed by using anesthetic ether and their stomachs were dissected out for determination of gastric lesions, washed in warm water and examined for ulcers microscopically with the help of hand lens (10x). Mean ulcer score for each animal in each group is expressed as ulcer index. Gastric juice collected into centrifuge tubes and centrifuged at 1000 rpm for 10 min and volume was noted. The p^H of the gastric juice was recorded by p^H meter and the gastric content is subjected for analysis of free and total acidity.

Statistical analysis

The values expressed as mean $\pm$ SD from 6 animals. The results were subjected to statistical analysis by using one way ANOVA followed by Dunnett's-'t'- test to verify the significant difference if any

among the groups. $P<0.05^*$, 0.01^{**} and 0.001^{***} were considered significant.

RESULTS:

Anti-ulcer activity:

Pylorus ligation induced ulcer model in rats:

In pylorus ligation induced ulcer model in rats a significant increase in ulcer number (5 ± 0.81), ulcer score (2.41 ± 0.27) and ulcer index (15.41) are noted. In the same model a significant increase in gastric volume (5.56 ± 0.54), free acid (6.8 ± 0.08 mEq/L) and total acid (12.61 ± 0.14 mEq/L) are noted.

Standard drug ranitidine 30 mg/kg treatment has significantly reduced ulcer number (0.16 ± 0.16) ulcer score (0.5 ± 0.12), ulcer index (4.32), gastric volume (4.35 ± 0.13 ml), free acid (2.78 ± 0.10 mEq/L) and total acid (7.75 ± 0.15 mEq/L).

In pylorus ligation induced ulcer model both AEFMC and AQEFMC except with low dose 100 mg/kg other two doses i.e, medium and high have significantly reduced the ulcer number (1.33 ± 0.33 , 0.83 ± 0.16 and 0.83 ± 0.10 , 0.75 ± 0.11) ulcer score (1.58 ± 0.08 , 1.41 ± 0.08 and 0.83 ± 0.10 , 0.75 ± 0.11) ulcer index (10.91, 14.28 and 8.15, 10.12) and the ulcer formation (25.84 %, 39.68 % and 53.18 %, 64.1 %) is significantly reduced.

Similar to the above a significant reduction in gastric volume (5.66 ± 0.35 , 5.55 ± 0.35 and 4.9 ± 0.29 , 5.38 ± 0.39 ml), free (5.15 ± 0.07 , 5.55 ± 0.10 and 3.06 ± 0.08 , 3.93 ± 0.08 mEq/L) and total acid (9.88 ± 0.31 , 9.56 ± 0.08 and 7.86 ± 0.12 , 8.46 ± 0.33 mEq/L) is noted with medium and high doses but not with the

low doses of AEFMC and AQEFMC respectively

Stress induced ulcer model in rats:

In stress induced ulcer model a significant increase in ulcer number (3.5 ± 0.71), ulcer score (2.25 ± 0.25) and ulcer index (17.75), gastric juice volume (5.8 ± 0.35 ml), free (8.01 ± 0.11 mEq/L) and total acids (14.43 ± 0.46 mEq/L) are noted.

Standard drug ranitidine 30 mg/kg treatment has significantly reduced ulcer number (0.5 ± 0.22), ulcer score (0.66 ± 0.10), ulcer index (5.16), gastric volume (5.45 ± 0.25), free acid (3.05 ± 0.27 mEq/L) and total acid (8.38 ± 0.40 mEq/L). Similar to pylorus ligation induced ulcer models AEFMC and AQEFMC except with low dose 100 mg/kg other two doses i.e, medium and high have significantly reduced the ulcer numbers (1.0 ± 0 , 1.0 ± 0 and 0.83 ± 0.16 , 0.66 ± 0.21) ulcer score (1.5 ± 0 , 1.33 ± 0.10 and 0.83 ± 0.10 , 0.75 ± 0.11) ulcer index (10.51, 2.33 and 7.99, 8.07) and the ulcer formation (20.63 %, 21.17 % and 36.57 %, 48.76 %) is significantly reduced.

So also a significant reduction in gastric volume (6.8 ± 0.41 , 7.81 ± 0.22 and 6.1 ± 0.10 , 6.31 ± 0.10 ml), free (5.65 ± 0.21 , 5.95 ± 0.14 and 6.1 ± 0.10 , 6.31 ± 0.10 mEq/L) and total acid (11.45 ± 0.44 , 12.26 ± 0.50 and 8.45 ± 0.25 , 9.4 ± 0.26 mEq/L) is noted with medium and high doses but not with the low dose of the extracts

Aspirin induced ulcer model in rats:

In aspirin induced ulcer model a significant rise in ulcer number (4.83 ± 0.54), ulcer score (2.5 ± 0.22), ulcer index (17.33), gastric volume (12.85 ± 0.16), free (8.46 ± 0.43 mEq/L) and total acids (16.3 ± 0.32 mEq/L) are noted.

Standard drug ranitidine 30 mg/kg treatment has significantly reduced ulcer number (0.33 ± 0.21), ulcer score (0.5 ± 0.12), ulcer index (4.13), gastric volume (4.5 ± 0.21 ml), free acid (2.78 ± 0.17 mEq/L) and total acid (9.26 ± 0.17 mEq/L).

Similar to the above two ulcer models both AEFMC and AQEFMC except with low dose 100 mg/kg other two doses i.e, medium and high have significantly reduced the ulcer numbers (2.16 ± 0.54 , 1.16 ± 0.40 and 0.66 ± 0.21 , 0.55 ± 0.22) ulcer score (1.83 ± 0.10 , 1.41 ± 0.51 and 0.58 ± 0.08 , $0.75 \pm 0.$) ulcer index (13.32, 11.90 and 5.9, 6.25) and the ulcer formation (20.63 %, 21.17 % and 36.57 %, 48.76 %) is significantly reduced. So also a significant reduction in gastric volume (6.46 ± 0.36 , 7.34 ± 0.34 and 5.15 ± 0.35 , 6.0 ± 0.30), free (4.65 ± 0.15 , 5.68 ± 0.46 and 3.38 ± 0.37 , 4.3 ± 0.17 mEq/L) and total acid (11.3 ± 0.40 , 12.0 ± 0.46 and 9.58 ± 0.29 , 10.5 ± 0.26 mEq/L) is noted with medium and high doses but not with the low doses of the both the extracts.

Table No: 5.33.Antiulcer effect of of AEFMC and AQEFMC in different ulcer models in rats

Group	Treatment	Pylorus ligation induced ulcers				Stress induced ulcers				Aspirin induced ulcers			
		Number	Score	Index	(%) Inhibition of ulcers	Number	score	Index	(%) Inhibition of ulcers	Number	score	Index	(%) Inhibition of ulcers
Control	Vehicle 10 ml/kg p.o	5± 0.81	2.41± 0.27	15.41	-	3.5± 0.71	2.25± 0.25	17.75	-	4.83± 0.54	2.5± 0.22	17.33	-
Standard	Ranitidine 30 mg/kg	0.16± 0.16**	0.5± 0.12**	4.32	86.67**	0.5± 0.22**	0.66± 0.10**	5.16	60.88**	0.33± 0.21**	0.5± 0.12**	4.13	76.16**
AEFMC	100 mg/kg p.o	3.33± 0.33 ns	2.16± 0.16 ^{ns}	13.49	11.02 ns	2.33± 0.61 ^{ns}	1.83± 0.29ns	12.16	8.95 ns	3.33± 0.33 ^{ns}	2.16± 0.16 ^{ns}	17.49	10.61ns
AEFMC	200 mg/kg p.o	1.33± 0.33*	1.58± 0.08 *	10.91	25.84*	1.00± 0**	1.5± 0*	10.5	20.63*	2.16± 0.54*	1.83± 0.10*	13.32	28.90*
AEFMC	400 mg/kg p.o	0.66± 0.21**	0.83± 0.10 **	8.15	53.18**	0.83± 0.16**	0.83± 0.10**	7.99	36.571**	0.66± 0.21**	0.58± 0.08**	5.9	54.41**
AQEFMC	100 mg/kg p.o	3.33± 0.66 ns	2.08± 0.20 ^{ns}	16.41	11.48 ns	1.66± 0.42	1.66± 0.10 ^{ns}	13.32	9.16 ns	3.0± 0.77ns	2.0± 0.22 ^{ns}	15.0	13.44 ns
AQEFMC	200 mg/kg p.o	0.83± 0.16*	1.41± 0.08 *	14.28	39.28*	1±0*	1.33± 0.10*	12.33	21.17*	1.16± 0.40*	1.41± 0.51*	11.90	37.10*
AQEFMC	400 mg/kg p.o	0.75± 0.11**	0.75± 0.11 **	10.12	64.1**	0.66± 0.21**	0.75± 0.11**	8.07	48.76**	0.55± 0.22**	0.75± 0.11**	6.25	63.93**

n = 6, Significant at P < 0.05*, 0.01** and 0.001***, ns = not significant

AEFMC-Alcoholic extract of fruit of *M.charantia* a AQEFMC- Aqueous extract of fruit of *M.charantia*

Fig No.5.31

Ant ulcer effect (% inhibition) of AEFMC and AQEFME in pylorus ligation induced ulcer model in rats

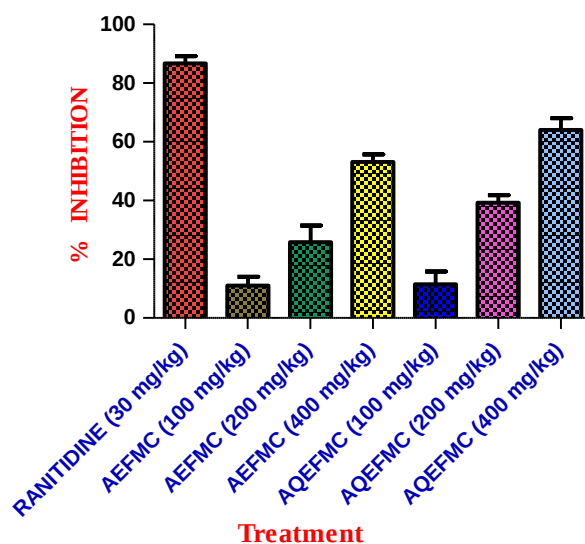


Fig No.5.32

Ant ulcer effect of AEFMC and AQEFME on Free acidity in pylorus ligation induced ulcer model in rats

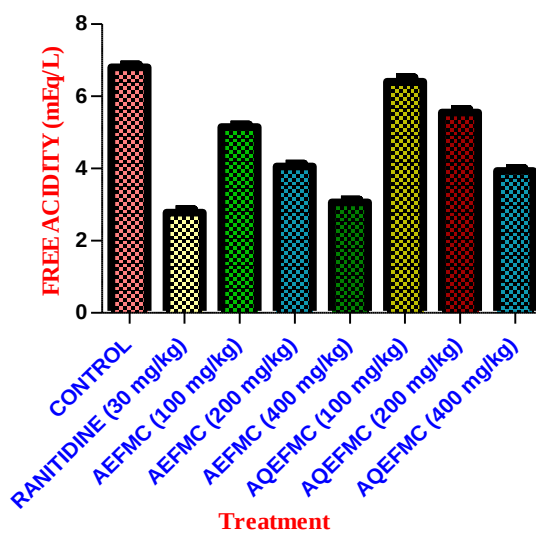


Fig No.5.33

Antiulcer effect of AEFMC and AQEFME on total acidity in pylorus ligation induced ulcer model in rats

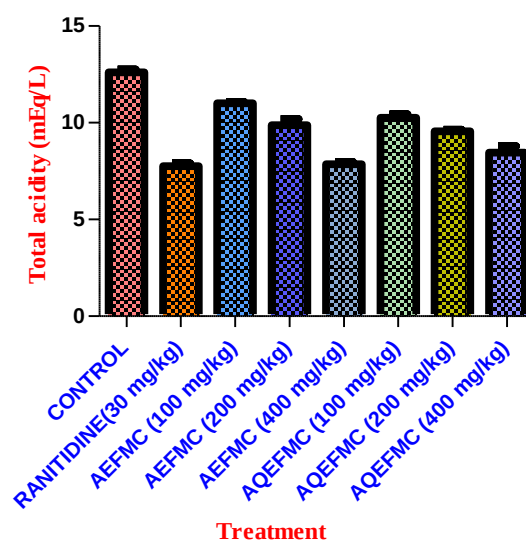


Fig No.5.34

Antiulcer effect of AEFMC and AQEFME on volume of gastric juice in pylorus ligation induced ulcer model in rats .

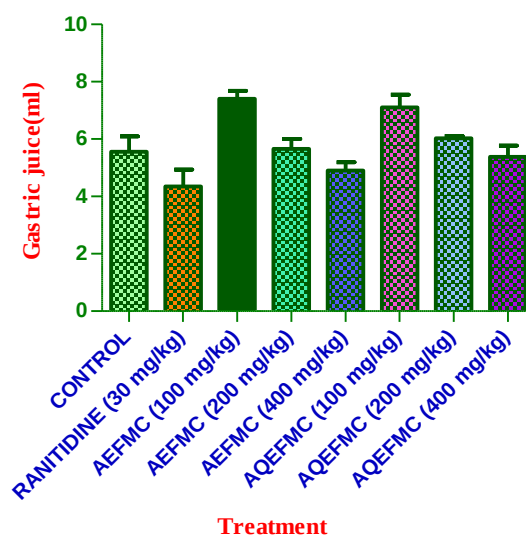


Fig No.5.35

Ant ulcer effect (% inhibition) of AEFMC and AQEFMC in stress induced ulcer model in rats .

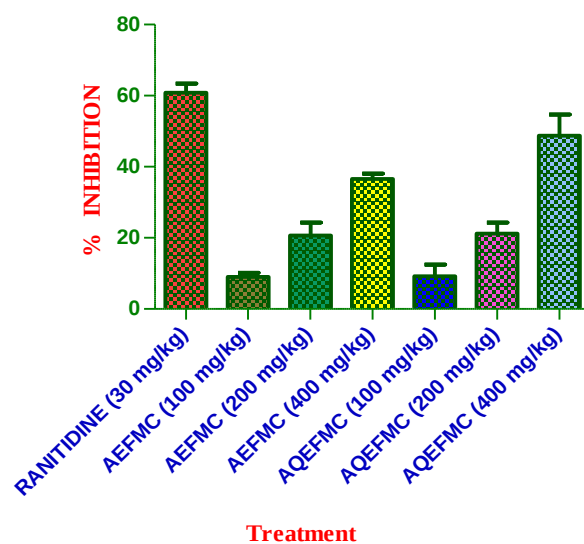


Fig No. 5.36

Ant ulcer effect of AEFMC and AQEFME on free acidity in stress induced ulcer model in rats .

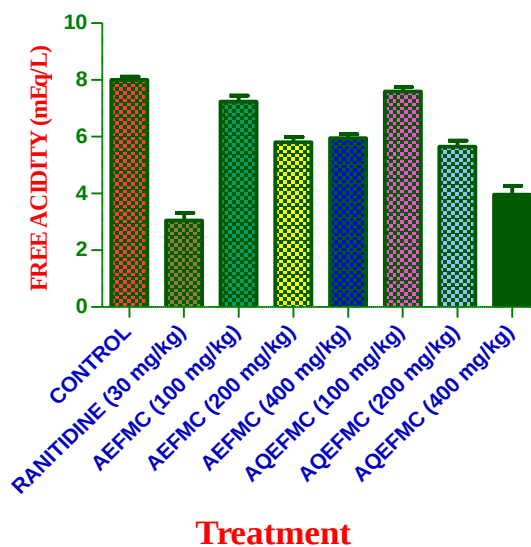


Fig No.5.37

Antiulcer effect of AEFMC and AQEFME on total acidity in stress induced ulcer model in rats .

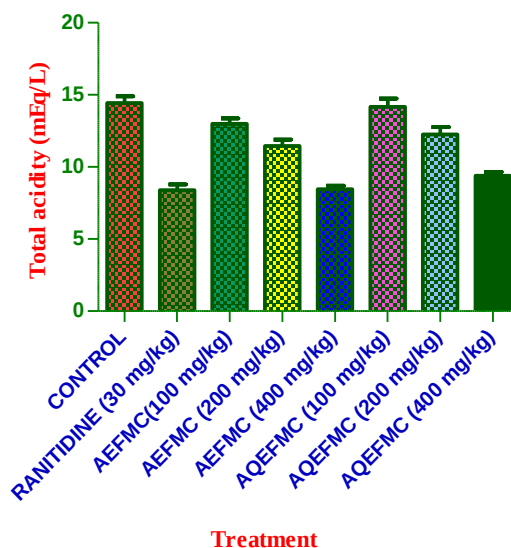


Fig No. 5.38

Antiulcer effect of AEFMC and AQEFME on volume of gastric juice in Stress induced ulcer model in rats .

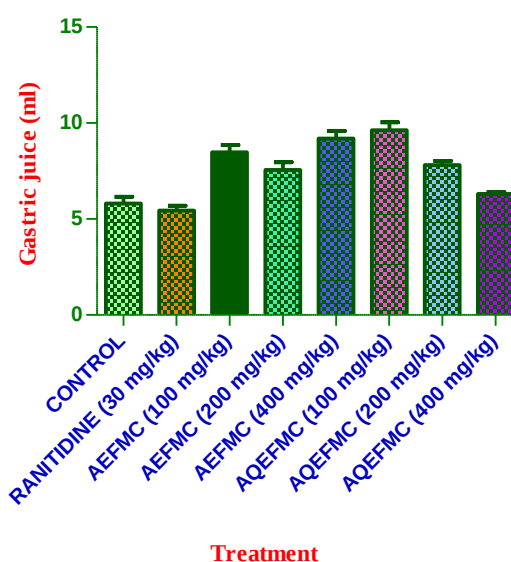


Fig No.5.39

Antiulcer effect (% inhibition) of AEFMC and AQEFME on Aspirin induced ulcer model in rats .

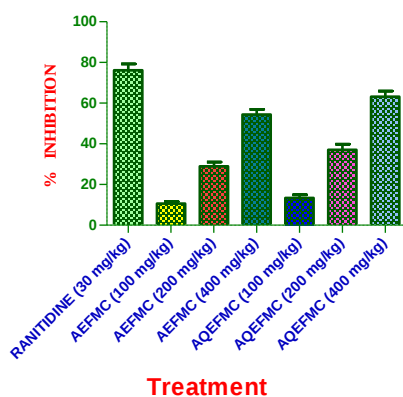


Fig No. 5.40

Antiulcer effect of AEFMC and AQEFME on Free acidity in stress induced ulcer model in rats .

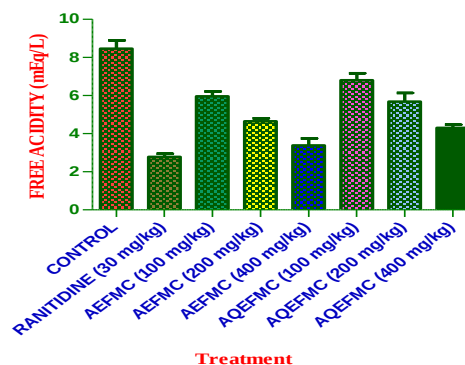


Fig No. 5.42

Antiulcer effect of AEFMC and AQEFMC on volume of gastric juice in Aspirin induced ulcer model in rats .

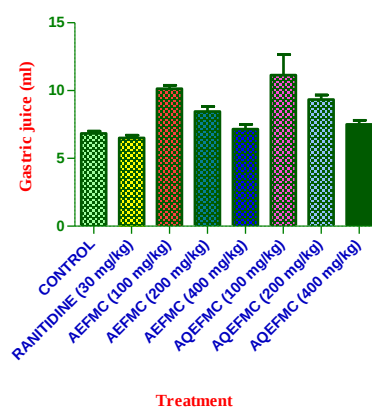
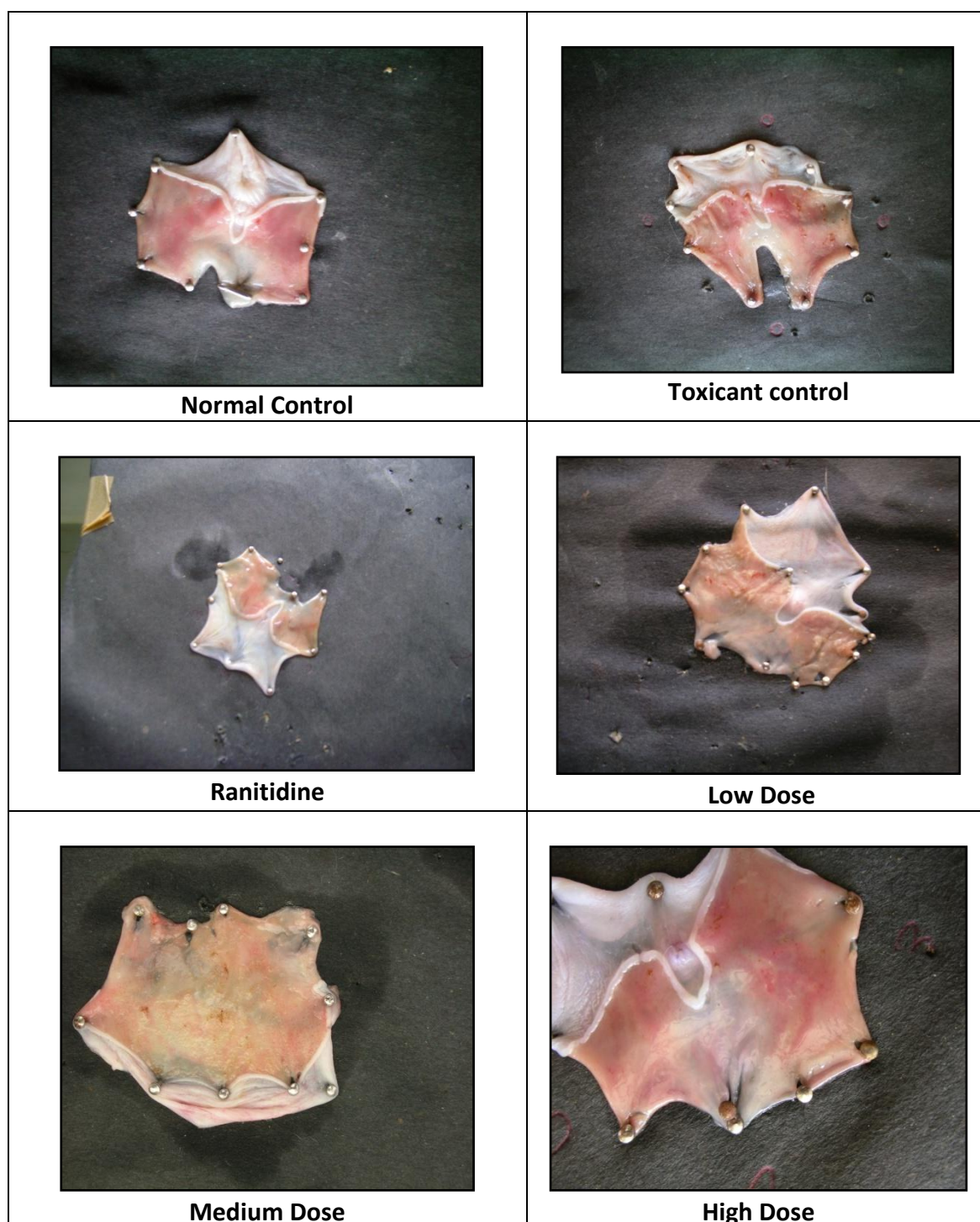


Figure: Effect of AEFMC on pylorus ligation induced ulcer model in rats



DISCUSSION

The fruit extracts (AEFMC and AQEFMC) of *M.charantia* were subjected for phytochemical investigation and LD₅₀ studies .It was found that alcoholic extract contained sterols, saponins, flavonoids, carbohydrates and alkaloids whereas aqueous extract contained saponins, flavonoids, carbohydrates and alkaloids. Both the extracts were tested for their lethal effect upto the dose level of 2000 mg/kg (up and down method) .None of them have produced abnormal behaviour or mortality in mice.

Three different doses namely low (1/5th), medium (1/10th) and high 1/20th) were selected with respect to the LD₅₀ dose i.e,2000 mg/kg b.wt for the present study.

Both the extracts were evaluated for their anti-arthritis activity in formaldehyde , Freund's adjuvant induced arthritis models in rats and Collagen induced arthritis model in mice. A significant (p<0.01) anti-arthritis activity was noted with both the extracts but relatively more activity with alcoholic extract which can be accounted for difference in phytoconstituents i.e,sterols as these were presented with alcoholic extract only.

Similarly both the extracts were evaluated for their anti-ulcer activity in pylorus ligation, stress and aspirin induced ulcer models in rats . Both the extracts produced a significant (p<0.01) anti-ulcer activity but similar to the above experiment a relatively better anti-ulcer activity was recorded with alcoholic extract.

CONCLUSION

Fruit extracts of *M.charantia* exhibited a significant anti-arthritis and anti-ulcer activities in experimental animals rats/mice.Alcoholic extract exhibited relatively better anti-arthritis and anti-ulcer activities than aqueous extract. The difference in the evaluated activities could be due to the number /quantity of phytoconstituents present in these extracts.

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