

## Moringa Oleifera Causes Gastro protection On Acid -Alcohol Induced Ulcer

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**Abstract:** Gastroprotective effects of *Moringa oleifera* was studied in 15 Wistar albino rats of both sexes weighing 350 - 450 g body weight (wt.). The rats were randomly allocated into 3 groups of 5 rats per group. Method for the preparation of animals and the surgical procedure were done according to the method of Ibu et al., 1986. Group 1 served as control (CTR), group 2 as acid alcohol (AA) alone and group 3 moringa + acid alcohol (MA) group. Results showed that moringa oleifera prevented ulceration when administered before acid alcohol. There was significant difference between group 1 and group 2; group 2 and group 3 ( $p < 0.01$ ). *Moringa oleifera* significantly offered gastroprotection against acid alcohol induced ulcer. It is concluded that moringa oleifera offers cytoprotection against acid-alcohol induced gastric ulcer

**Key works** (*Moringa oleifera*, gastroprotection, gastric ulcer)

Date of Submission: 30-06-2018

Date Of Acceptance: 13-07-2018

### I. Introduction

The gastrointestinal system has many unusual functions, but none more remarkable than the production of highly acidic juice by the stomach. It is phenomenal that the stomach should secrete a juice not vital to body economy, but a juice which is intimately involved in a common illness [1]. Peptic ulcer can only occur in the presence of gastric acids which mean "No acid, no ulcer" [2]. The pathophysiology of peptic ulcer disease involves an imbalance between offensive (acid, pepsin, and *Helicobacter pylori*) and defensive factors such as mucin, prostaglandin, bicarbonate, nitric oxide, and growth factors [3].

Alcohol consumption is considered to be a risk factor in the development of gastric and duodenal ulcers. Ethanol rapidly penetrates the gastrointestinal mucosa, causing membrane damage, exfoliation of cells and erosion. The subsequent increase in mucosal permeability together with the vasoactive products from mast cells, macrophages and blood cells may lead to vascular injury, necrosis, generation of free radicals and ulcer formation [4]. It also generates superoxide anion ( $O_2^-$ ) and hydroxyl radical ( $\cdot OH$ ) which induces ulcer in experimental animals [4].

Although many products are used for the treatment of gastric ulcers e.g. antacids and antihistaminics. Most of these drugs, however, produce several side effect [5]. Extracts of many herbal plants have been shown to produce promising results for the treatment of gastric ulcer and *Moringa oleifera* is one of them [6].

*Moringa Oleifera* (*Moringaceae*) is a plant grown world-wide. [7]. Almost all parts of this plant -root, leaves, fruit, bark, seeds, and flowers are reported to have important medicinal values as cardiac and circulatory stimulants and to have antitumor, antipyretic, antiinflammatory, antihypertensive, diuretic, cholesterol lowering, antidiabetic, antioxidant, antispasmodic, antibacterial, and antifungal activities [8]. Based on this numerous documented effects of *moringa oleifera*, this study was carried out to elucidate if moringa has any effect on other action alcohol on

### II. Materials and Method

Fresh leaves of *Moringa oleifera* were collected from the natural habitat in Makurdi, Benue state, Nigeria. Sample of leaves collected were identified by a taxonomist from the Department of Botany in the Faculty of Science, Benue State University and were allocated a voucher number and deposited in the herbarium of the department.



Figure 1: Fresh leaves of *Moringa oleifera*

## 2.1 PREPARATION OF EXTRACT

The leaves were sorted out to obtain only the fresh leaves and washed with distilled water without squeezing to remove debris and dust particles. They were shade dried for ten days and dried leaves pulverized with electric blender. A portion (300 g) of the powdered leaves were soaked in 1500ml of distilled water for 72hours with the solution thoroughly stirred twice daily according to the method of Onahinon et al [9]. The extract was filtered with WHATMAN no1 filter paper. The filtrate was dried using evaporator and then reconstituted before use

## 2.2 CHEMICALS

Urethane (ethylcarbamate), HCl and Ethanol made by Sigma chemical Co. (Poole, UK). Marketed by Sigma Aldrich-USA. These were purchased from EMOLE chemical shop, old Otukpo road High Level Makurdi, Benue State Nigeria

## 2.3 ANIMALS

Adult Wistar albino rats weighing 300-450g of either sex were purchased from the disease-free stock of the animal house of the College of Health Sciences, Benue State University, Makurdi and used for the study. They were maintained in normal and standard laboratory conditions of temperature 28°C and relative humidity (with 12-hour light dark cycle) and adequate ventilation. The animals were fed with commercial diet (Vital Feed Nig. Ltd.) and water *ad libitum*. Food was withheld 12 hours before the experiments, but they had free access to water. Permission for the use of animals and animal house were obtained from the Animal Ethics Committee of Benue State University Makurdi, prior to experimentation.

## 2.4 SURGICAL PROCEDURE AND INDUCTION OF ULCER

After a 12 hour fast, each animal was anaesthetized with 25 % Ethyl Carbonate (urethane) at a dose of 0.6 ml /100 g body weight intraperitoneally. Tracheostomy was performed. A nasogastric tube was passed. A duodenostomy was performed and normal saline was used as gastric lavage to wash out the debris from the stomach until clear effluent was obtained. A duodenogastric canula was passed and ligated insitu for subsequent collection of gastric acid secretion Measurement of gastric acid secretion was done according to method of Gosh and Schild [9] as modified by Ibu [10]. Basal acid secretions was collected for the first 2 hours and titrated every 10 minutes for each hour. *Moringa oleifera* leaf aqueous extract was administered one hour before acid alcohol was given. The aliquots were collected every 10mins for 120mins (2hrs). Each aliquot was titrated to a phenolphthalein end point using 0.01M NaOH and the acid output or concentration was calculated as described by Ibu [10] as follows:

Where Normality = Molarity

$$MA \times VA = MB \times VB \dots\dots\dots(1)$$

$$MA = (MB \times VB) \div VA \dots\dots\dots(2)$$

Where,

MB = Molarity of base known (0.01N) = 10mMol

VB = Volume of base known (titrate of NaOH) used

VA = Volume of acid (effluent volume) = 10ml

Substituting for MB and VA

$$MA = 10 \times VB \div 10 \dots\dots\dots (3)$$

$$\text{Therefore } MA=VB \dots\dots\dots (4)$$

$$\text{Acid output / 10 minutes} = VB \text{ mMol / L / 10 mins} \dots\dots\dots (5)$$

$$\text{Acid output per hour} = VB \times 6 \text{ mMol/ L / hour as stated by Ibu [10]} \dots\dots\dots (6)$$

**2.5 Animal grouping.**

The animals were randomly allocated into three (3) groups of 5 animals per group (n=5) as follows:

1. Control group: Non-Ulcer induced (administered normal saline orally) 5 -10mls in 6 hours. [10].
2. Acid Alcohol group: (36% HCL 0.25 ml) + 75% Ethanol 0.25ml/100g body weight [11].
3. *Moringa oleifera* group : 50mg /100 g body weights given 1hr before acid Alcohol (36% HCL (0.25 ml) + 75% Ethanol (0.25 ml) / 100 g body weights [12].

**2.6 DATA ANALYSIS**

Data obtained from the study was expressed as mean ± SEM. The differences between the groups were analysed by one-way analysis of variance (ANOVA), followed by Turkey's post Hoc test for multiple comparisons using SPSS statistical tool version 22.

**III. Results**

**Table 1**

Time	Control (mmol/L/hr.)	Acid alcohol (alch.) (mmol/L/hr.)	Moringa + acid alch. (mmol/L/hr.)
10 mins	8.16±0.05	9.60±0.06	4.44±0.02
20 mins	8.52±0.04	10.2±0.03	4.32±0.02
30 mins	8.16±0.05	12.1±0.07	3.84±0.02
40 mins	8.28±0.02	12.7±0.07	3.60±0.00
50 mins	8.28±0.02	14.5±0.13	3.36±0.02
60 mins	8.40±0.00	16.2±0.10	3.48±0.02

Table 1 showed the Gastric Acid secretions (mmol/L/hr) of various groups taken every 10 minutes, one hour after acid alcohol was given.

**TABLE 2**

Time	Control (mmol/L/hr.)	Acid alcohol (mmol/L/hr.)	Moringa+ acid alch. (mmol/L/hr.)
10 mins	8.40±0.05	12.8±0.04	1.32±0.02
20 mins	8.52±0.04	13.3±0.06	0.60±0.00
30 mins	8.64±0.02	14.3±0.05	0.60±0.00
40 mins	8.76±0.02	14.2±0.11	0.60±0.00
50 mins	8.76±0.02	15.0±0.03	0.60±0.00
60 mins	8.76±0.02	15.5±0.02	0.6±0.00

Table 2 showed the Gastric Acid secretions (mmol/L/hr) of various groups taken every 10 minutes, 2 hours after acid alcohol was given

**Table 3**

Wistar rats	Control group	Acid alcohol alone group	Moringa + acid alcohol group
Rat 1	0	4	0
Rat 2	0	3	0
Rat 3	0	4	0
Rat 4	0	4	0
Rat 5	0	3	0
Total	0	22	0
Ulcer index	0	3.60±0.3	0±0

Table 3 is ulcer score and ulcer index of the control group, acid alcohol group, Moringa + acid alcohol group

TABLE 4 ANOVA

ulcer score					
	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	43.200	2	21.600	216.000	.000
Within Groups	1.200	12	.100		
Total	44.400	14			

Table 4 is the analysis of variance (ANOVA) of the ulcer score. It showed that there was significant difference between groups (P<0.01)

TABLE 5

ulcer score				
	groups	N	Subset for alpha = 0.05	
			1	2
Tukey HSD <sup>a</sup>	1	5	.00	
	3	5	.00	
	2	5		3.60
	Sig.		1.000	1.000

Means for groups in homogeneous subsets are displayed.  
a. Uses Harmonic Mean Sample Size = 5.000.

Table 5 is Turkey Homogenous subsets of the ulcer score of the contol in group 1, acid alcohol with normal saline in group2 and *moringa oleifera* given before acid alcohol in group 3. There was no statistical significant difference between group 1 and group 3 (p<0.01) but there was significant difference between group 1 and group 2, group 2 and group 3 (p>0.01).

Fig. 2 Bar chart with 95% confidence level of gastric acid secretions (mmol/L/hr) in control, acid alcohol alone and Moringa + acid alcohol groups after 1 hour

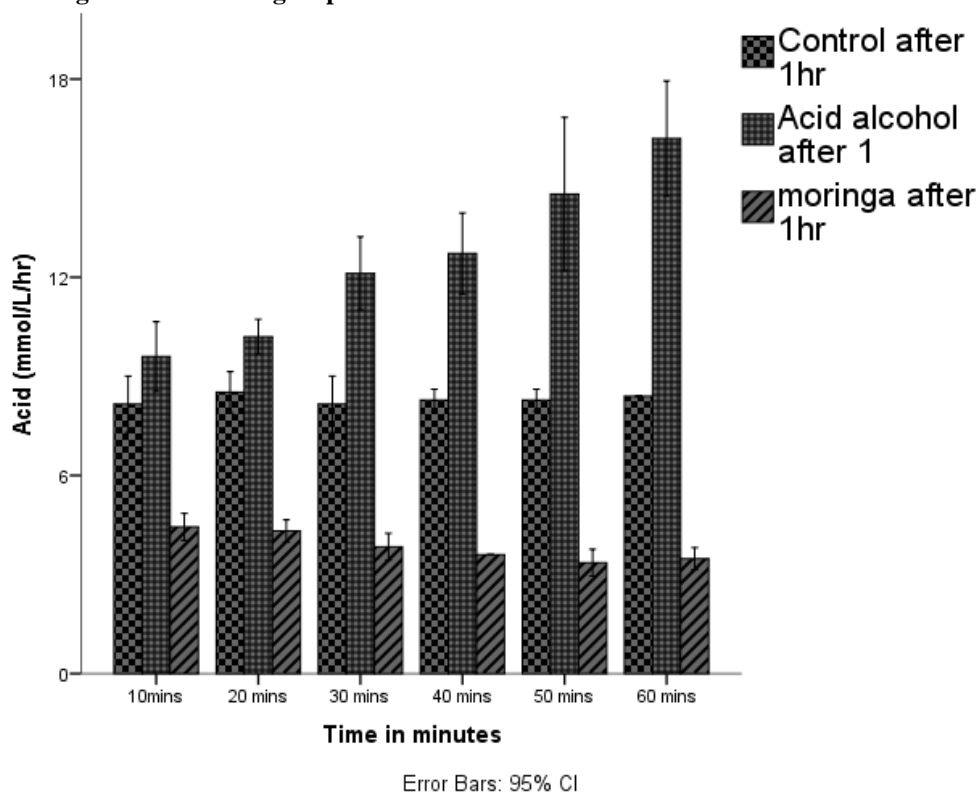


FIGURE 2 showed that there was time dependent increase in gastric acid secretion after the administration of acid alcohol. Fig 2 also showed that *moringa oleifera* significantly reduced gastric acid secretion (p<0.01)

**Fig. 3** Bar chart with 95% confidence level of acid secretions (mmol/hr) of control, acid alcohol alone and Moringa + acid alcohol groups after 2 hours

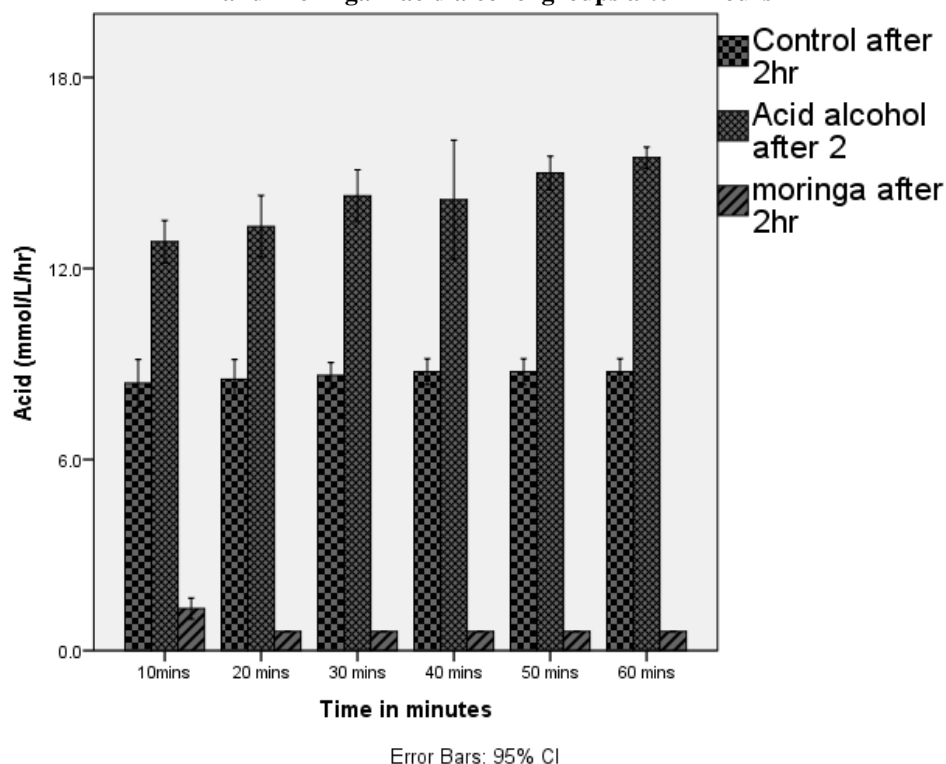


FIGURE 3 showed contol and the effect of acid alcohol and *moringa oleifera* in the 2 hour after administration

#### IV. Discussion

The result obtained from this study showed that acid alcohol significantly increased gastric acid secretion ( $p < 0.01$ ), this agrees with other work earlier done [14, 15, 16]. Acid alcohol easily penetrates the gastric mucosa and causes gastric ulcer [16] one hour after administration in rats [14]. Gastric ulcers induced by acid alcohol are not only associated with a decrease in gastric mucus, but also an increase in lipid peroxidation, oxidative stress inside the cells, changes in permeability and depolarization of the mitochondrial membrane, which ultimately leads to cell and membrane damage [16]. Acid alcohol administration produces hemorrhagic lesions, infiltrated inflammatory cells, extensive submucosal edema, epithelial cell loss and mucosal friability in the stomach which are typical symptoms of alcohol injury [17]. The result from this study also showed that *Moringa oleifera* significantly protect the gastric mucosa from acid alcohol induced lesions ( $p < 0.01$ ) this agrees with previous studies [18, 19, 20]. The gastric protective effects of *Moringa oleifera* aqueous extracts may be due to its direct action on the mucus secretion or by increasing prostaglandins, thus protecting the stomach from acid alcohol injury. It has also been reported to alter the antioxidant factors like total tissue sulfhydryl group (glutathione) suggesting that the prevention of the development of gastric ulcers in rats by *Moringa oleifera* is due to its antioxidant action [21]. The gastro-protective and antioxidant effects of *Moringa oleifera* is as a results of it several active constituents such as alkaloids, sterols, glycosides, flavonoids, and terpenoids [22]. Also, its leaves are rich in benzyl isothiocyanate which has anti-inflammatory activity [23]. It has also been reported that antioxidant properties of *M. oleifera* leaf extracts exert its action via alteration in superoxide dismutase (SOD), glutathione peroxidase (GPx), and malondialdehyde (MDA) levels in rat gastric mucosa [24]. In gastric ulcer condition, there is an increase in gastric mucosal SOD and lipid peroxidation activities which shown that the generation of reactive oxygen species (ROS) such as superoxide anion ( $O_2^-$ ),  $H_2O_2$  and hydroxyl radical ( $OH^\cdot$ ) induce cell degeneration by increasing lipid peroxidation of cell membrane lipids and might be the causative factor for the inactivation of gastric peroxidase. Therefore, antioxidant effects of *M. oleifera* leaf extracts is probably by metabolizing lipid peroxides and scavenging endogenous hydrogen peroxide ( $H_2O_2$ ) [25]. Hessah, 2018 [16] demonstrated that *Moringa oleifera* can restore the antioxidant activities of GST, GPx, and SOD and decrease the lipid peroxidation induced by oral administration of acid alcohol and with notable decrease in gastric lesions. Therefore, the gastro protective effect of *Moringa oleifera* found in this study is as a result of its anti inflammatory and anti oxidant activity against acid alcohol induced ulcer.

## V. Conclusion and Recommendation

*Moringa oleifera* significantly offers gastroprotection against acid alcohol induced ulcer. *Moringa oleifera* can therefore be used as prophylactic against mucosa lesion induced by alcohol. Thus, it is recommended that Health Education of the populace on the importance of this findings should be initiated.

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Ankita Jangde "Self Ligating Brackets From Past To Present: An Update." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, vol. 17, no. 7, 2018, pp 44-49.