Alhagi Maurorum Methanol Extract ameliorates gastric ulceration and liver injury in a rat model

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ABSTRACT

Methanol extract was prepared from *Alhagi maurorum* and evaluated for its potential antiulcerogenic and hepatoprotective effects in rats. Preliminary phytochemical analysis and acute LD_{50} were also carried out. Oral administration of the methanol extract (400 and 800 mg kg^{-1}) of *Alhagi maurorum* significantly ($P<0.05$) decreased the average ulcer index in a dose dependent manner with a curative ratio of 69.6 and 74.6% for the small and large dose respectively, in the ethanol-induced gastric ulceration. Based on the decreased ulcer index, *Alhagi maurorum* induced significant antiulcerogenic effect against aspirin-induced gastric ulceration with a curative ratio of 90.3 and 85.6% respectively. The number of ulcers and total acidity were significantly ($P<0.01$) decreased. Improvement of the histopathological picture of liver tissue and the serum biochemical parameters (AST, ALT, GGT and Albumin) indicated that a good hepatoprotective effect against CCl_4-induced hepatotoxicity was achieved by oral administration of methanol extract of *Alhagi maurorum.* No symptoms of discomfort were reported after doses of up to 2.5 g kg^{-1}. Methanol extract of *Alhagi maurorum* was reported to contain unsaturated sterols, triterpenes, tannins, flavonoids and carbohydrates and/or glycosides as active constituents. GC-Ms analysis revealed the presence of 24 compounds of which Luteolin (14.88%), Amarogentin (23.22%), Palmitic acid (24.23%), inoleic acid (6.6%) are major constituents. This study proved that the methanol extract of *Alhagi maurorum* has a promising gastro- and hepatoprotective effect in rats.

Keywords: Antiulcer, hepatoprotective, *Alhagi maurorum*, GC/MS, phytochemical.

INTRODUCTION

Although there are large number of products that have been used as antiulcerogenic, and hepatoprotective, there are no completely effective drugs that stimulate hepatic function, offer complete protection to the organ, or aid in regenerating hepatic cells (Chattopadhyay 2003). Additionally, some drugs can induce adverse or side effects (Shirani et al., 2017). Therefore, there is a need for potent and less toxic antiulcerogenic and hepatoprotective agents. Plant extracts are

the most attractive sources of drugs since long time ago. A number of plants have been proved to produce a broad range of biological activities such as antioxidant (Laghari et al., 2012), anti-ulcer, antispasmodic, antidiarrheal (Atta and Mouneir, 2004), antinociceptive (Atta and Abo El-Sooud, 2004), antipyretic (Alam et al, 2016), promising antiulcerogenic and hepatoprotective effects (Alkofahi and Atta 1999, Singh et al, 2012) *Alhagi maurorum* (*A. maurorum*) is a shrub native to temperate, tropical and Mediterranean region but has been introduced to many other areas of the world, including Russia, Australia, southern Africa, northern India and the western United States. The hepatoprotective protective effect of *Alhagi maurorum* (*A. maurorum*) was tested in rabbits intoxicated with paracetamol (Rehman et al., 2015). The traditional uses and the pharmacological actions of *Alhagi species* are reviewed (Ahmed et al., 2015, Asghari et al., 2016). In addition, Alhagi plants are also a good source of digestible protein and important minerals (Muhammad et al., 2015). People customarily use the extracts of this plant for their gastrointestinal sedative effects without any scientific base. For this reason, we studied the antiulcerogenic effect of *A. maurorum* commonly grown in Upper Egypt and in the River Nile and Delta regions against acute and prolonged gastric mucosal damage models. Moreover, the hepatoprotective effect of methanol extract of the plant against carbon tetrachloride-induced hepatotoxicity in rats was also studied.

MATERIAL AND METHODS

1. Plant material

A. maurorum collected from River Nile and Delta regions was identified by the staff members of the Herbarium of the Department of Botany, Faculty of Science, Cairo University. A voucher sample was kept in the Department of Pharmacology, Faculty of Veterinary Medicine, Cairo University, Egypt. The air-dried plant was moderately pulverized and stored in dark bottles. The powdered plant (200 g) was extracted with methanol 95% for at least 24 h, followed by percolation for 5 to 7 times till complete exhaustion. The methanol extracts was concentrated under reduced pressure using Rotary Evaporator at temperature not more than 50 °C. The concentrated extract was kept at –4 °C until used for phytochemical and pharmacological studies.

2. Antiulcerogenic effect

2.1. Ethanol induced gastric ulceration

Twenty male Sprague-Dawley rats (150-200 g b.wt.) were kept under standard conditions before use. Rats were randomly divided into 4 equal groups. Animals were starved for 48 h before use to ensure an empty stomach and were kept in cages with raised floors of wide wire mesh to prevent coprophagy (Garg et al., 1993). To prevent excessive dehydration during the fasting period rats were supplied with sucrose (BDH) 8% (w/v) solution in NaCl (BDH) 0.2% (w/v) which was removed 1 h before experiments (Glavin and Mikhail, 1976).

In the first day, rats of groups one and group two were orally given two doses of each of 400 and 800 mg kg^{-1} b.wt. of *A. maurorum* extract suspended in 2% Tween 80 with 6 h apart. A third dose was given in the 2nd day 1.5 h before oral administration of ethanol (Merck) 50% (v/v in distilled water) in a dose of 10 ml kg^{-1} b.wt. A control group was given equal volume of 2% Tween 80 in distilled water instead of the plant extracts but received ethanol in the same dose and route. In addition a fourth group was given ranitidine as a reference drug at a dose of 100 mg kg^{-1} b.wt. by the same route and at the same time intervals. One hour after ethanol administration, all rats were euthanized by an over dose of chloroform and the stomachs were rapidly removed, opened along their greater curvature and the long lesions were counted and measured and the petechial lesions were counted as described by Ogle et al., (1985). The ulcer index (mm) and the curative ratio were calculated as described by the following formula (Cho and Ogle, 1978).

Curative ratio = (Control ulcer index – Test ulcer index)/ (Control ulcer index) x 100

2.2. Aspirin induced ulceration

Twenty male Sprague-Dawley rats (150-200 g b.wt.) kept under standard conditions were randomly divided into 4 equal groups. The modified method of Goel et al. (1985) was used for the production of experimental gastric ulceration. Aspirin (200 mg kg^{-1}) suspended in carboxymethyl cellulose 1% was administered orally. Two doses of methanol extract of *A. maurorum* (400 and 800 mg kg^{-1}) were given 3 h prior to and after aspirin administration. Treatment continued for 3 days and the pylorus was ligated on the fourth day. The abdomen was opened under ether anaesthesia and the pylorus was legated with silk suture. The abdomen was closed and the animals were left to recover and drinking water was withheld for 4 h

after which rats were killed with an overdose of chloroform, the oesophagus was legated and the stomach was removed. The gastric mucosa was washed with 3 ml distilled water. The gastric juice and the washings were collected and centrifuged at 5000 rpm for 5 min. The volume of gastric juice was measured and expressed as ml/100 g b.wt. The stomach was opened and the glandular portion was examined, the number of ulcers was counted and the total length was measured. The curative ratio was calculated as mentioned before. A third and fourth group were given distilled water or ranitidine (the reference drug) instead of the plant extract and rats were dissected as previously described.

2. 3. Determination of the total gastric acid output

The method described by Goyal et al. (2004)[18] was used. One ml of the gastric juice in 10 ml of distilled water was titrated with 0.01 N NaOH using phenolphthalein as an indicator. Data were expressed as mEq/ml of the gastric juice.

2. 4. Determination of total proteins content in the gastric juice

Biuret Reagents was applied to determine the total proteins in the gastric juice (Anoop and Jegadeesan, 2003) [19] using commercial kits.

3. Hepatoprotective effect

3.1. Animals

Five groups of 5 rats each were used. First group (normal control) was given 2.5ml of corn oil kg^{-1} b.wt. daily for 5 days. Rats of the second group were given the same dose of corn oil and on the 5th day, CCl_4 (50% in corn oil) were given in a single oral dose of 2.5 $ml.kg^{-1}$ b.wt. 2 h after the last dose of the oil. The third and fourth groups were pre-treated orally with 400 and 800 mg kg^{-1} b.wt. of the Alhagi methanol extract daily for 5 days and CCl_4 was given 2 h after the last dose. Moreover, rats of the fifth group were treated with 25 mg kg^{-1} b.wt. silymarin (reference drug) daily for 5 days and 2 h after the last dose, CCl_4 was given as before. Blood samples were collected by puncturing of the tail vein 24 h after the hepatotoxin administration. Blood samples were placed in plain centrifuge tubes for serum separation. Serum was

used for determination of the activities of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (Reitman and Frankel, 1957), gamma glutamyl transferase (GGT) (Szasz, 1969), glucose (Sokoloff et al., 1977), total bilirubin(Westwood, 1991), triglycerides (Fossati and Prencipe,1982), total cholesterol (Allain et al., 1974), total proteins (Burtis et al., 2014) and albumin (Doumas et al., 1971).

3.2. Histopathological studies

Liver tissue specimens were collected from all rats immediately after thiopental 1% intravenous euthanasia and fixed in 10% formol saline. Paraffin sections of 5µ thickness were prepared, stained by H & E and examined microscopically (Suvarna et., 2018).

4. Acute toxicity study in mice

The acute toxicity of plant extract was tested using three doses (0.4, 1 and 2.5 g kg^{-1} b.wt., orally) as described by Tanira et al. (1996) but the observation period extended up to 72 h post administration. In addition, the general behaviour of animals, signs of discomfort, or any nervous manifestations were observed.

5. Phytochemical screening

5.1. Preliminary phytochemical screening

The extracts were screened for the presence of tannins, saponins, sterols/ triterpenes, alkaloids, anthraquinones, flavonoids, lactones/esters, protein/amino acids and carbohydrates and/or glycosides with thin layer chromatography (TLC) as described by Wall (2007). Detection of anthraquinones, saponins, tannins, glycosides/ carbohydrate are carried out using KOH, anisaldehyde-sulphuric acid, ferric chloride and naphthoresorcinol reagents respectively (Wagner et al., 1983). Detection was carried out by visualization in visible light and under UV light (366 nm).

5. 2. GC-Mass analysis of methanol extract

Gas Chromatograph (GC, Agilent Technologies 7890A) interfaced with a mass-selective detector (Agilent 7000 Triple Quad) was used. Agilent HP-5ms capillary

column (30 m×0.25 mm ID and 0.25 µm film thicknesses) was used. The flow rate was 1 mL/min. The injector and detector temperatures were 200 ^{O}C and 250 ^{O}C, respectively. The acquisition mass range was 50-600. The formulae of the components as identified by comparing their mass spectra and RT with those of NIST and WILEY library were recorded.

6. Statistical analysis

Results are expressed as mean ± standard deviation (SD). Differences between control and treated groups were tested for significance using a one-way analysis of variance (ANOVA). *P* values of 0.05 or less were considered significant.

RESULTS

1. Antiulcerogenic Effects

1.1. Alcohol induced ulcers

Oral administration of methanol extract of *A. maurorum* induced a dose dependant decrease ($P<0.05$) in the alcohol-ulcer index with a curative ratio of 69.6 and 74.60% following a dose of 400 and 800 mg kg^{-1}, respectively. This is compared to 26.1% for ranitidine, the reference drug (Table 1).

1.2. Aspirin induced ulcers

The methanol extract of *A. maurorum* significantly ($P<0.5$) reduced the gastric ulcer index in aspirin -induced gastric ulceration. The number of long ulcers and total gastric acidity was significantly ($P< 0.01$) decreased. The volume of gastric juice and the total protein concentration in gastric juice was not significantly affected. Ranitidine significantly ($P<0.05$) decreased the ulcer index, the number of ulcers and the total acidity but did not affect the volume of gastric juice and the total proteins content (Table 2).

2. Hepatoprotective effect

CCl_4 significantly (P<0.05) elevated the activity of serum liver enzymes (AST, ALT, GGT). *A. maurorum* methanol extract significantly decreased the activity of these enzymes to values comparable to rantidine, particularly when used in the large dose (Table 3). Serum glucose and albumin were significantly (P<0.05) decreased in CCl_4-intoxicated animals but there was No significant change in total proteins, globulins or A/G ratio. *A. maurorum* methanol restored the level of glucose and albumin in a dose dependent manner (Table 4). CCl_4 significantly (P<0.05) decreased serum triglycerides level but significantly increased serum bilirubin concentration. *A. maurorum* methanol extract elevated triglycerides and decreased total bilirubin and cholesterol levels (Table 5).

3. Histopathological examination

Cross sections in liver of control rats showed normal hepatic architecture of the hepatocytes arranged in hepatic cords around the central veins (**Figure** 1a). Oral administration of CCl_4 into rats induced severe degenerated and necrobiotic changes (centrolobular) in the hepatocytes associated with severe fatty change and infiltration of mononuclear inflammatory cells surrounding the central vein as well as in focal manner between the hepatocytes (Figure 1b). Liver of rats pre-treated with *A. maurorum* methanol extract at a dose 400 mg kg^{-1} then intoxicated CCl_4 showed moderate degenerated and necrobiotic changes in the centrolobular area of the hepatic lobules, with moderate fatty change in the hepatocytes and mononuclear inflammatory cells infiltration in between (**Figure** 1c). Mild degenerative and necrobiotic changes in the hepatocytes with mononuclear inflammatory cells infiltration in between were seen in the livers of CCl_4–intoxicated rats pre-treated with *A. maurorum* methanol extracts at a doses 800 mg kg^{-1} (**Figure** 1d). Liver of rats pre-treated with silymarin then intoxicated CCl_4 showed moderate degenerative and necrobiotic centrolobular changes in the hepatocytes with mononuclear inflammatory cells infiltration in between (**Figure** 1e).

4. Acute toxicity study in mice

Oral administration of *A. maurorum* methanol extract in doses up 2.5 g kg^{-1} b.wt. did not cause any major signs of acute toxicity. No deaths were reported up to 72 h after oral administration.

5. Phytochemical screening of A. maurorum methanol extract

Preliminary phytochemical screening of *A. maurorum* extract revealed the presence of unsaturated sterols, triterpenes, tannins, flavonoids and carbohydrates and/or glycosides. No alkaloids, anthraquinones, lactones/esters, protein/amino acids or saponins were found.

GC-MS analysis revealed that the methanol extract contains 24 compounds of which 14 compounds of more than 1% (Figure 2 and Table 6). The major components (>1%) are L-Glucose (5.53%), Ouabagenin (4.32%), D-Xylose (1.56%), 3',4'-Dihydroxyflavone (1.85%), d-Mannose (4.7%), Luteolin (14.88%), Amarogentin (23.22%), Phytol (2.73%), Palmitic acid (24.23%), Deoxyestradiol (1.36%), Chromone, 5-hydroxy-6,7,8-trimethoxy-2,3-dimethyl-(1.38%), Linoleic acid (6.6%), Stearic acid (1.41%), trans-Phytoene (1.34%).

DISCUSSION

The present data demonstrate the efficacy of *A. maurorum* methanol extract against gastric ulceration induced by two experimental models; acute ethanol- and prolonged aspirin-induced gastric ulcerations. The mechanism by which ethanol can induce gastric ulceration is direct nonspecific necrotic effects (Oates and Hakkinen, 1988). However, activation of TNF alpha expression followed by apoptosis in the gastric mucosa and consequently an increase in the severity of ulcerative damage in the stomach has been proposed (Liu and Cho, 2000). Reactive oxygen species can be involved in the pathogenesis of these ulcers (Khosla et al., 2004). On the other hand, the use of non-steroidal anti-inflammatory drugs is prohibited by gastric mucosal irritation and ulcer formation. Gastric acid and pepsin secretion, suppression of gastric microcirculation, prostaglandin E2 inhibition (Laine et al., 2008), and pro-inflammatory cytokines interleukin (IL)-1β and tumour necrosis factor (TNF)-α activation (Appleyard et al., 1996) are important factors in the development of gastric ulcers (Wang et al., 2013).

A. maurorum methanol extract, although, decreased the number of ulcers, total acidity and increased curative ratio but it did not decrease the volume of gastric juice indicating that *A. maurorum* methanol extract acts as cytoprotective rather than anti-secretory. Similar conclusion has been reported to other plant extracts (Deshpande et al., 2003). Phytochemical screening revealed that *A. maurorum* methanol extract contains variable amounts of triterpens and flavonoids which

appeared to be extracted by methanol (Akhtar et al., 1992) (Akhtar and Ahmad, 1995). GC/MS analysis revealed that the methanol extract of *A. maurorum* contains high percent of Luteolin (a flavonoid flavone) and Amarogentin. Luteolin is the major component of *Markhamia tomentosa* which exert a potent gastric antiulcer effect (Sofidiya et al., 2014). Amarogentin exerts potent selective cyclooxygenase-2 inhibition (Shukla et al., 2014). Moreover it has an in vitro and in vivo antitumor effects against human gastric cancer cells (Zhang et al., 2010) and exhibit potential immunomodulatory effects (Lad and Bhatnagar 2016). Triterpens are the active constituents that have been claimed to be effective as anti-ulcer agents because they protect the mucosa against acid effects by selective inhibition of prostaglandins F2 alpha (De Pasquale et al., 1995). The methanol extract of *A. maurorum* contains also kaempferol, which have a wide range of pharmacological activities, including antioxidant, anti-inflammatory, antimicrobial, anticancer, cardioprotective, neuroprotective, antidiabetic, anti-osteoporotic, estrogenic/ antiestrogenic, anxiolytic, analgesic and antiallergic activities (Calderon-Montano et al., 2011). Moreover, the flavonoids have been proved to possess antiulcerogenic effect (Mota et al., 2009), most likely because of inhibition of eicosanoid generating enzymes or the modulation of the production of pro-inflammatory molecules (Liu and Cho, 2000) and their antioxidant effect (Bubols et al., 2013). Recent studies have also shown that some flavonoids are modulators of pro-inflammatory gene expression, thus leading to the attenuation of the inflammatory response(Leyva-López et al., 2016). Carbon tetrachloride administration to experimental animals induced acute pathological changes in the liver. The biochemical mechanism of CCl_4 intoxication could involve the generation of toxic metabolites of carbon tetrachloride which have been implicated to be the cause of hepatocellular damage. It has been shown that liver microsomal oxidizing enzymes connected with cytochrome P-450 produce reactive metabolites of CCl_4 such as trichloromethyl radical (CCl_3) or trichloroperoxyl ($CCl3O_3$). These radicals cause lipid peroxidation, disturbance in calcium (Ca^{2+}) homeostasis and finally cell death (Recknagel et al., 1989). The toxic metabolites increase lipid peroxidation (Day, 2002), reduce protein synthesis, decrease serum levels of secreted albumin, decrease the rate of triacylglycerol secretion and initiate the inhibition of lipoprotein secretion and thus steatosis (Boll, 2001). Damage of hepatocytes cause the release of cytoplasmic enzymes and increase serum aminotransferases (ALT, AST) and GGT activities (Giannini et al., 2005).

The active constituents of methanol extract of *A. maurorum* (flavonoids, carotenoids) are probably responsible for the protective effect of plant extract against CCl4-induced toxicity (Gilani and Janbaz 1995). Triterpenoids and steroids among the plant constituents that possess hepatoprotective effect (Morikawa et al., 2014) because of their potential antioxdant effect (Grassmann et al., 2005) (Upadhyaya et al., 2009). Flavonoids are known for their antioxidants, free radical scavengers and antilipoperoxidants leading to hepatoprotection (Al-Qarawi et al., 2004). Carotenoids are also known to induce antioxidants and antihepatotoxic

activity (Fiedor and Burda 2014). Unsaturated sterols, triterpenes, tannins, flavonoids and carbohydrates and/or glycosides are reported as the active compounds of *A. maurorum* which may exert beneficial protective action by toxin-mediated lipid peroxidation either via decreased production of carbon tetrachloride derived free radicals or through their direct antioxidant activity (Olas et al., 2015). Moreover, GC/MS analysis revealed that the methanol extract of *A. maurorum* contains high percent of Amarogentin. It has been reported that amarogentin from *Swertia patens* (Gentianaceae) exert significant hepatoprotective effects against carbon tetrachloride-induced liver fibrosis in mice (Dai et al., 2018) and anti-inflammatory effect (Shukla et., 2014).

CONCLUSION

The present results confirm the traditional use of *A. maurorum* for treatment of gastrointestinal disturbances. Additional experiments are required to isolate and test the active constituents to explore the exact mechanism of action of these plants.

ACKNOWLEDGEMENT

The Authors extend their appreciation to Cairo University for financial support.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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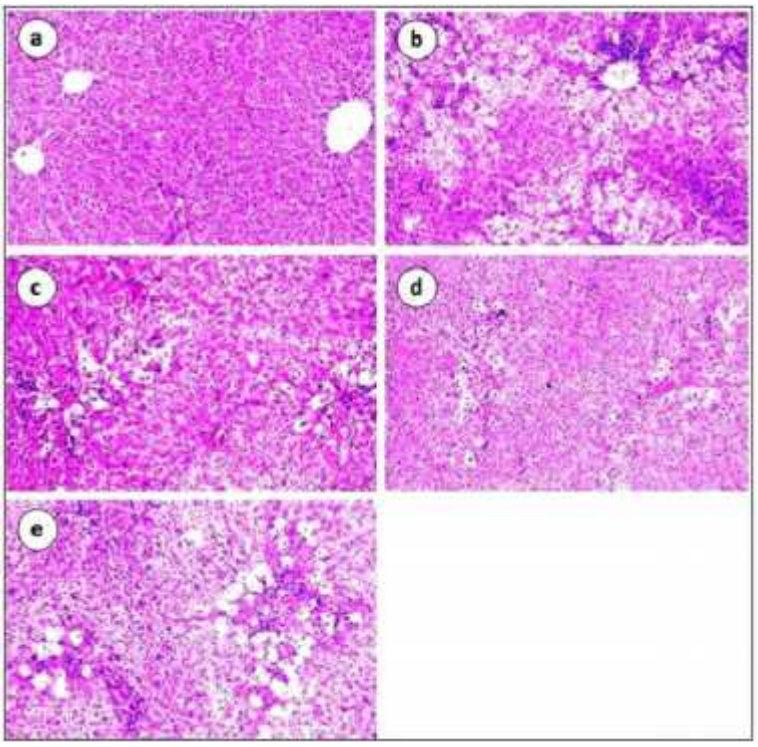

Figure 1: Histopathological sections in liver of (a) normal control rat showing normal histological structure of the hepatocytes in the hepatic cords with central veins, (b) CCl_4- intoxicated rat, (c) CCl_4-intoxicated rat treated with methanol extract of *A. maurorum* (400 mg kg^{-1}), (d) CCl_4-intoxicated rat treated with methanol extract of *A. maurorum* (800 mg kg^{-1}) and (e) CCl_4-intoxicated rat treated with silymarin. (H&E ×40)

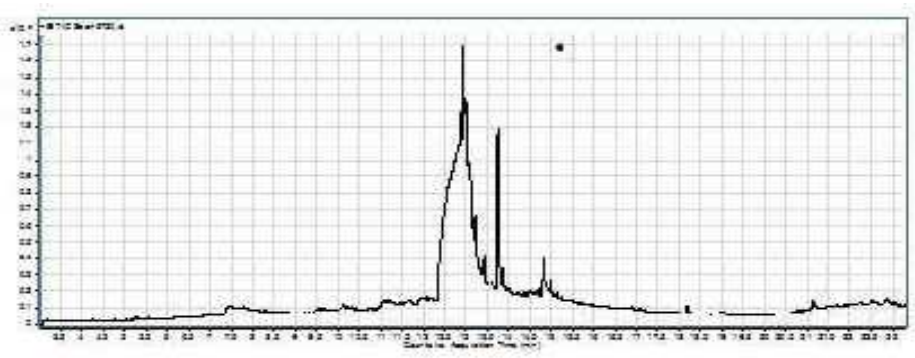

Figure 2: GC-MS peaks of *Alhagi maurorum* methanol extract.

Table 1: Effect of *Alhagi maurorum* methanol extract (400 and 800 mg.kg^{-1} b. wt.) on ethanol induced gastric ulceration in rats.

Groups	Ulcer index	Curative Ratio
Control	41.40±5.80	-
A. maurorum 400	12.60±3.40*	69.60
A. maurorum 800	10.50±1.03*	74.60
Ranitidine 100 mg.kg^{-1}	30.60±3.80*	26.10

* Significant at $P<0.05$.

Table 2: Effect of *Alhagi maurorum* methanol extract (400 and 800 mg.kg^{-1} b. wt.) on aspirin induced-gastric ulceration in rats (mean ± SD, n=5)

Groups	Ulcer index	Curative ratio%	Number of ulcers	Volume of gastric juice (ml/100g b.wt.)	Total acidity (mEq/l)	Total proteins (µg/ml)
Control	14.40±4.60	0.00	18.11±2.58	3.10±0.50	37.00±4.80	0.84±0.20
A. maurorum 400	1.40±0.30**	90.30	6.33±0.58*	3.30±0.70	10.80±2.80**	0.74±0.15
A. maurorum 800	2.08±0.39**	85.60	0.70±0.20**	2.90±0.80	7.65±1.80**	0.85±0.25
Ranitidine 100 mg.kg^{-1}	7.40±1.40**	48.60	3.20±0.80**	3.00±0.60	2.88±0.70**	0.80±0.16

* Significant at $P<0.05$. ** Significant at $P< 0.01$.

Table 3: Effect of *Alhagi maurorum* methanol extract (400 and 800 mg.kg^{-1} b. wt.) on some serum liver enzyme activities of rats (Mean ± SD, n=5).

Groups	AST (U/l)	ALT (U/l)	GGT (U/l)
Control	209.8±20.13^{a}	76.20±7.09^{a}	7.63±1.64^{a}
CCl4	738.00±51.67^{c}	474.00±40.79^{b}	21.60±5.94^{b}
A. maurorum400	282.00±28.64^{a}	115.20±17.58^{a}	8.08±1.58^{a}
*A. maurorum*800	262.80±34.27^{a}	108.40±15.13^{a}	6.54±0.73^{a}
Ranitidine100 mg.kg^{-1}	390.00±49.50^{b}	152.20±28.06^{a}	7.20±1.10^{a}

Means with different asterisk letters are significantly different at $P<0.05$.

Table 4: Effect of *Alhagi maurorum* methanol extract (400 and 800 mg.kg^{-1} b. wt.) on blood glucose, total proteins and its fractions of rats (Mean ± SD, n=5).

Groups	Glucose (mg/dl)	Total proteins (g/dl)	Albumin (g/dl)	Globulin (g/dl)	A/G Ratio
Control	126.2±6.0^{c}	6.86±1.1^{a}	4.04±0.3^{ab}	2.74±0.5^{a}	1.52±0.3^{a}
CCl4	89.0±8.0^{a}	6.22±0.4^{a}	3.26±0.4^{a}	2.94±0.4^{a}	1.18±0.2^{a}
A. maurorum400	102.0±9.1^{b}	6.54±1.1^{a}	3.46±0.4^{a}	2.84±0.2^{a}	1.15±0.2^{a}
*A. maurorum*800	126.2±6.5^{c}	7.16±1.1^{a}	5.18±0.9^{c}	2.84±0.6^{a}	1.09±0.5^{a}
Ranitidine100 mg.kg^{-1}	108.4±13.3^{b}	7.44±0.8^{a}	4.58±1.1^{bc}	3.10±0.5^{a}	1.48±0.3^{a}

Means with different asterisk letters are significantly different at $P<0.05$.

Table 5: Effect of *Alhagi maurorum* methanol extract (400 and 800 mg.kg^{-1} b. wt.) on serum triglycerides, cholesterol and total bilirubin of rats (mean ± SD)

Group	Triglycerides (mg/dl)	Cholesterol (mg/dl)	Total bilirubin (mg/dl)
Control	141.60±8.2^{d}	83.60±6.5^{b}	0.34±0.03^{b}
CCl4	76.00±21.6^{ab}	77.40±6.4^{b}	0.42±0.07^{c}
A. maurorum400	80.00±7.1b^{c}	63.00±13.2^{a}	0.28±0.03^{a}
*A. maurorum*800	85.98±9.4^{c}	59.00±10.2^{a}	0.35±0.03^{b}
Ranitidine100 mg.kg^{-1}	68.00±8.3^{a}	63.00±9.1^{a}	0.36±0.05b^{c}

Means with different asterisk letters are significantly different at $P<0.05$.

Table 6: GC-MS analysis of *Alhagi maurorum* methanol extract.

	Rt	Name	Area sum%
1.	5.27	2,3-Dehydro-4-oxo-β-ionol	0.79
2.	5.55	8-Carboxy-3-methylflavone	0.86
3.	7.5	L-Glucose	5.53
4.	9.54	Ouabagenin	4.32
5.	10.12	Quercetin 3',4',7-trimethyl ether	0.6
6.	10.3	all trans-Retinal	0.59
7.	11.08	D-Xylose	1.56
8.	11.68	3',4'-Dihydroxyflavone	1.85
9.	12.87	d-Mannose	4.7
10.	12.921	Luteolin	14.88
11.	12.97	Amarogentin	23.22
12.	13.2	Phytol	2.73
13.	13.78	Palmitic acid	24.23
14.	13.83	Deoxyestradiol	1.36
15.	13.99	Isomyristic acid	0.26
16.	14.18	8-Carboxy-3-methylflavone	0.24
17.	14.522	Chromone, 5-hydroxy-6,7,8-trimethoxy-2,3-dimethyl-	1.38
18.	14.69	Kampferol-3,4'-dimethyl ether	0.47
19.	14.83	Linoleic acid	6.6
20.	14.98	Stearic acid	1.41
21.	15.12	Arachidic acid	0.36
22.	15.25	Salicylic acid β-D-O-glucuronide	0.27
23.	16.3	Sclerotiorin	0.44
24.	22.54	trans-Phytoene	1.34